A COMPARATIVE STUDY OF OPEN SURGICAL DRAINAGE AND EARLY PERCUTANEOUS NEEDLE ASPIRATION OF LIVER ABSCESS.

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M. S. General Surgery

Branch – I

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The Tamilnadu Dr. M. G. R. Medical University

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CERTIFICATE

Certified that this dissertation entitled **"A COMPARATIVE STUDY OF OPEN SURGICAL DRAINAGE AND EARLY PERCUTANEOUS NEEDLE ASPIRATION OF LIVER ABSCESS"** is a bonafide work done by **Dr. C. R. SIDDARTHA**, post graduate student of the Department of General Surgery, Chengalpattu Medical College, during the academic year 2010 – 2013. This work has not previously formed the basis for the award of any degree.

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I, **Dr. C. R. SIDDARTHA,** solemnly declare that dissertation titled, "**A COMPARATIVE STUDY OF OPEN SURGICAL DRAINAGE AND EARLY PERCUTANEOUS NEEDLE ASPIRATION OF LIVER ABSCESS"** is a bonafide work done by me at Chengalpattu Medical College during 2010-2013 under the guidance and supervision of **Prof. Dr. G. RAJA BILLY GRAHAM ,** Professor and Head, Department of General Surgery, Chengalpattu Medical College.

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Introduction

Liver Abscess noted in the writings of Hippocrates, was considered to be dreadful disease. It was uniformly fatal until a study by Ochsner and Debakey in 1938. They suggested an aggressive surgical approach to the liver abscess which reduced the mortality rates considerably. [1] Advances in radiological imaging which can diagnose the presence of liver abscess and its accurate anatomical localisation, combined with advances in pharmacotherapeutics and critical care have shifted the treatment modality of choice from open surgical drainage to minimally invasive procedures. With many therapeutic options now available, the effective treatment of liver abscess is still controversial. Do the minimally invasive procedures provide effective drainage of the abscess with reduction of mortality when compared with open surgical drainage? This study aims at comparing the two methods of abscess drainage, open surgical and percutaneous aspiration in their effectiveness of providing cure in liver abscess.

Review of literature

History

Liver abscess has been known since the time of Hippocrates (460 - 377 BC). In one of his aphorisms he mentions “when abscess of the liver is treated by the cautery or incision, if the pus which is discharged is 'pure and white', the patient recovers, but if it resembles the lees of the oil as it flows, they die” .[2]

Around 300 BC, Alexander the Great, after his campaign reaching the Indus region, where amoebiasis is endemic, felt sick and died on his return in Gedrosia desert. It is believed that is death was probably due to amoebic liver abscess. [3]

In 1818, George Ballingall, an army surgeon at the Madras establishment had made a note of an officer who got accidentally shot, resulted in draining of liver abscess and obtaining a complete cure. [4]

Napoleon Bonaparte, the French emperor in 18th century was exiled to a tropical island St. Helena after his defeat at the Battle of Waterloo. He had contracted a disease which was common on the island. Antommarchi, a physician appointed by Napoleon’s family described the features of Napoleon’s last illness. At the emperor's post-mortem, he had noticed “a cancerous ulcer which had its centre at the superior part along the small

curve of stomach, communicating with the liver." It is believed that Napoleon had died of amoebic liver abscess of the left lobe which had ruptured into stomach. [3]

In 1887, Robert Koch investigating cholera in Egypt and India had come across 2 cases of dysentery complicated by liver abscess. He identified Entamoeba histolytica in the capillary walls, adjacent to the abscess and found them similar to the ones in stool. [3]

Sir Charles Morehead, Professor of Medicine at Grants Medical College, Bombay in 1948, had described the first case of hepatic abscess in India. [3]

In 1891, Councilmann and Lafleur in their monograph coined the term "Amoebic Abscess of the liver" and described the occurrence of liver abscess as a complication of intestinal amoebiasis even in patients who did not have the symptoms of latter disease. [5]

Sir William Osler, in his 1892 edition of "The Principles and Practice of Medicine" described the major cause of liver abscess as "suppuration in the territory of portal vessels". He also stated that "results from dysentery and other ulcerative affections of the bowels, appendicitis, occasionally typhoid fever, in rectal affections, and in abscesses in the pelvis." He termed the process of portal bacteraemia with abscess formations as "suppurative pylephlebitis". [6]

Ochsner and DeBakey in 1938 described 137 cases of amoebic abscess and 47 cases of pyogenic abscess. They also described the aggressive surgical management to decrease mortality which was a uniformly fatal disease until then. [1] Mcfadzean et al in 1953 first described the successful treatment of pyogenic liver abscess by percutaneous aspiration. [7] Whereas in 2005, a study by Tan et al showed open surgical drainage maybe a better modality compared to percutaneous drainage for the treatment of liver abscess. [8] Numerous studies have been published and yet effective treatment of liver abscess remains controversial.

Epidemiology

Liver abscess is a rare occurrence whose aetiology, diagnosis and treatment have changed over time. Traditionally, it has been classified into pyogenic abscess – caused by various bacteria, and Amoebic – caused by Entamoeba histolytica. Recent trends show increase in the incidence of fungal and mycobacterial abscess, probably due to increase in patients with AIDS and other forms of immunosuppression. [9]

Liver abscesses are the most common type of visceral abscess; in a report of 540 cases of intra-abdominal abscesses, pyogenic liver abscesses accounted for 48 percent of visceral abscesses and 13 percent of intra-abdominal abscesses. [10] The annual incidence of liver abscess has been estimated at 2.3 cases per 100,000 and is higher among men than women.

[11] Risk factors include diabetes, hepatobiliary or pancreatic disease, and post liver transplantation. A primary invasive liver abscess syndrome due to Klebsiella pneumoniae has been described in East Asia indicating interplay between host and geographical factors. [12] With the effective treatment of conditions like appendicitis and other acute colonic diseases, there has been a shift in the aetiology and age distribution of patients presenting with hepatic abscess. Numerous studies have reported biliary tract disease to be the most frequent underlying lesion associated with pyogenic liver abscess, with a peak incidence in the seventh and eight decades. [11]

Worldwide, amoebiasis is the third most common parasitic cause of death. [13] Amoebic liver abscess is 7 to 10 times more common among adult men, despite equal gender distribution of intestinal amoebiasis. It is predominantly seen in the fourth and fifth decades of life. The reasons for these observations are not fully understood; suggested mechanisms include hormonal effects and a potential role of alcoholic hepatocellular damage in creating a nidus for portal seeding. Immigrants and travellers from endemic regions, people of low socioeconomic status, mentally retarded individuals and male homosexuals have a predilection for amoebic liver abscess. [9]

Pyogenic Liver Abscess

Aetiology

Liver is constantly exposed to the infective organisms from the gastrointestinal tract. Kupffer cells act as a filter for the clearance of micro organisms. Abscesses occur when normal hepatic clearance mechanism fail or the system is overwhelmed.

The potential cause of liver abscess is classified as [14]:

1. Biliary.
2. Portal vein.
3. Haematogenous.
4. Traumatic.
5. Direct extension.
6. Cryptogenic.

30 – 40% of all pyogenic abscesses are due to diseases of the biliary system. 40% of these abscesses occur due to underlying malignancy. Obstruction of biliary tree, intrahepatic biliary stone, associated stricture, manipulation of biliary tract like cholangiography, percutaneous hepatic stents, endoscopic stent placement and biliary enteric anastomosis predispose patients to cholangitis and pyogenic liver abscess. [14]

Intestinal pathology is responsible for 20% of pyogenic liver abscess. Gastrointestinal perforation or bacterial translocation cause transient bacteraemia and resultant spread via the portal vein to the liver. Diverticulitis, perforated colonic carcinoma and other intra-abdominal abscess predispose to pylephlebitis and cause liver abscess. [14]

Gangrenous cholecystitis, perforated ulcers and subphrenic abscesses cause liver abscess by contiguous spread. Trauma to liver causes parenchymal necrosis and clot, which creates an ideal milieu for the seeding and proliferation of microorganisms and subsequent abscess formation. Haematogenous spread can occur from distant infection from heart, lungs, kidneys, bones, ears and teeth through the hepatic artery and cause about 12% of pyogenic liver abscesses. Cryptogenic abscesses, those of unknown aetiology occur in 10 – 45% of patients who have co morbidities like diabetes, immunosuppression. [14]

Pathology

The number, size, location of liver abscess is determined by the source. Portal, cryptogenic, and traumatic abscesses are solitary, large whereas biliary and arterial abscesses are small and multiple. [14]

Huang et al reported that liver abscess involved the right lobe in 63% of cases; the left lobe in 14% and 22% had bilobar disease. [11] The

predilection for the right hepatic lobe can be attributed to anatomic considerations. [14]

- The right hepatic lobe receives blood from both the superior mesenteric and portal veins, whereas the left hepatic lobe receives inferior mesenteric and splenic drainage.
- The right lobe contains a denser network of biliary canaliculi and, overall, accounts for more hepatic mass.
- Studies have suggested that a streaming effect in the portal circulation is causative.

Bacteriology

Abscess cultures are positive for growth in 80 – 90% of cases, but blood cultures yield growth in 50 – 60%. E. coli, klebsiella sp, enterococci, pseudomonas are commonly isolated aerobic bacteria. [15] Bacteroides and fusobacterium are the commonly isolated anaerobes. [16]

The organisms commonly isolated from the abscess cavity and their frequency of isolation is described in the table below.

Category of organisms	% of patients
Gram negative aerobes	50 – 70
Eschericihia coli	35 – 45
Klebsiella	18
Proteus	10
Enterobacter	15
Serratia	Rare
Morganella	Rare
Acinetobacter	Rare
Gram positive aerobes	55
Streptococcal species	20
Enterococcus	10
β – Streptococci	5
α - Streptococci	5
Staphylococcal sp	15
Anaerobes	40 – 50
Bacteroides	24
Fusobacterium	10
Peptostreptococcus	10
Clostridium	5
Actinomyces	Rare
Fungal	26
Sterile	7

Clinical features

The clinical presentation of liver abscess is usually sub-acute and nonspecific, requiring high index of suspicion for diagnosis. The classic presentation triad of fever with chills, right upper quadrant pain and general malaise is rarely present. [15] The most common presenting symptom is fever with chills. The abdominal pain is constant dull aching, localised to the right upper quadrant. Associated diarrhoea is rarely present. Abscesses of the left lobe present with symptoms in the epigastric region. Associated features like malaise and weight loss are present when patients present late. Jaundice occurs when there is compression of the biliary tree. Pleuritic pain or pain at the tip of right shoulder occurs due to irritation of the right hemi-diaphragm. [14]

The following table shows the common signs and symptoms of presentation of patients with pyogenic liver abscess and their frequency of occurrence.

Symptom	Percentage of pyogenic abscesses
Fever	83
Weight loss	60
Pain	55
Nausea and vomiting	50
Malaise	50
Chills	37
Anorexia	34
Cough or pleurisy	30
Pruritis	17
Diarrhoea	12
Signs	
Right upper quadrant tenderness	52
Hepatomegaly	40
Jaundice	31
Right upper quadrant mass	25
Ascites	25
Pleural rub or effusion	20

Complications

Complications of liver pyogenic liver abscess can be classified into

1. Local complications include:
    - Rupture [17]:
        - Into the peritoneal cavity resulting in peritonitis.
        - Into the pleural cavity causing empyema and hepatopleural fistula.
        - Into the pericardial cavity from an abscess of the left lobe.
        - Into adjacent organ and fistulise into stomach, colon, small bowel, or kidney.
        - Subphrenic space resulting in subphrenic space.
        - Into biliary tract resulting in haemobilia.
    - Liver Failure.
    - Acute pancreatitis.
2. Vascular complications include:
        - Hepatic vein thrombosis.
        - Portal vein thrombosis.
        - Hepatic artery pseudo – aneurysm due to erosion of abscess into the hepatic artery.

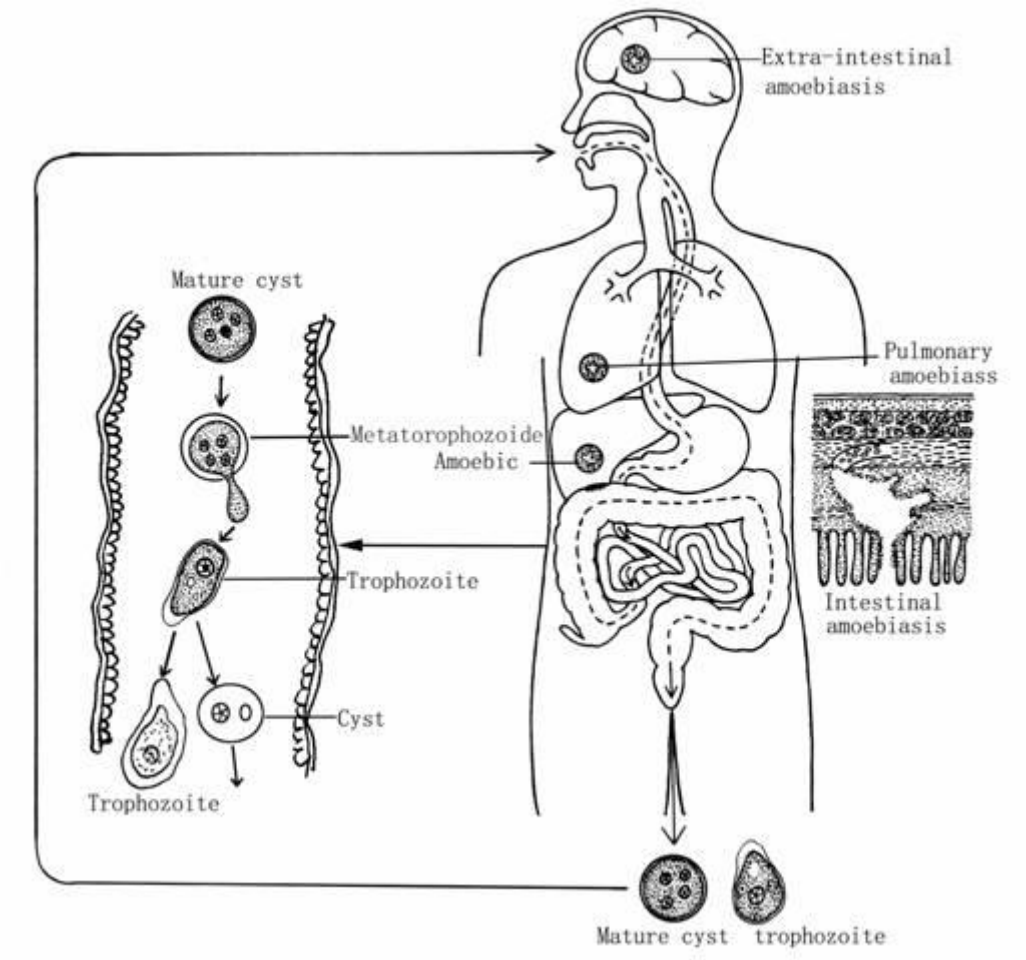
3. Systemic septic complications are common in diabetic patients who are infected with Klebsiella pneumonia through haematogenous dissemination. [18]
    - Endophthalmitis or uveitis,
    - Pulmonary abscess,
    - Brain abscess and/or purulent meningitis,
    - Bacteriuria and/or prostate abscess,
    - Osteomyelitis and/or pyogenic arthritis, and
    - Psoas abscess.

Amoebic liver abscess

Aetiology

Amoebic liver abscess is caused by Entamoeba histolytica. After fecal oral transmission, the infective quadrinucleate cyst passes through the stomach. The trophozoite is released after the pancreatic enzymes digest the outer cyst wall. Usually, the trophozoite multiplies in the intestine with no invasion resulting in amoebic dysentery alone or asymptomatic carrier. In a few patients, the trophozoite invades the intestine and travels along the veins and reaches the liver and form an abscess cavity. [14]

Life cycle of Entamoeba histolytica



Pathology

Entamoeba histolytica as implied by its name lyses through the tissues through a complex set of interactions, cell adherence, cell activation and subsequent release of enzymes results in cell necrosis. Hepatic amoebic abscess more commonly involves the right lobe of liver. It is a result of

liquefactive necrosis of the liver forming a cavity of blood and liquefied liver tissue. The hepatic necrosis continues until it reaches the Glisson's capsule. As the capsule is resistant to hydrolysis, the amoebic liver abscess tends to abut the liver capsule and the cavity is crisscrossed by portal triads. [19]

Early on, the infection of liver results in amoebic hepatitis which progress to from multiple small abscess which later coalesce to form a single large hepatic abscess. The abscess itself contains acellular fluid which is red/brown and yellow in colour and similar to “anchovy paste” in consistency. Trophozoites are absent in the abscess but reside in the necrotic tissue surrounding the abscess. [20]

Clinical presentation

Majority of amoebic liver abscess occur in the young adult males. The presentation may be acute, with fever and right upper quadrant pain, or sub acute, with weight loss. History of alcohol abuse is common.

The following table shows the common signs and symptoms of presentation of patients with pyogenic liver abscess and their frequency of occurrence.

Symptom	Percentage of Amoebic liver abscesses
Pain	90
Fever	87
Nausea and vomiting	85
Anorexia	50
Weight loss	45
Malaise	25
Diarrhoea	25
Cough or pleurisy	25
Pruritis	<1
Signs	
Hepatomegaly	85
Right upper quadrant tenderness	84
Pleural effusion or rub	40
Right upper quadrant mass	12
Ascites	10
Jaundice	5

Complications

- Secondary bacterial infection of the abscess can occur.
- Pleural manifestations may include development of a sympathetic serous effusion.
- Rupture is the most common complication of amoebic liver abscess. Rupture may occur into peritoneum, pleural cavity or pericardium, the latter being common with left lobe abscess. Rupture may also occur into the stomach and colon. Abscess rupture into the pleural space results in an amoebic empyema; rupture into the lung can lead to consolidation, abscess formation, or a hepatobronchial fistula resulting in a spontaneous cure. [21]
- Metastatic amoebic abscess can occur in brain, lung, skin and genitourinary tract, presumably due to haematogenous spread from the intestinal infection. [21]
- Compression syndrome: A posterior located Amoebic liver abscess of the right lobe may manifest as hepatic outflow obstruction or inferior vena cava obstruction. Clinical features include bilateral pedal oedema, ascites and visible veins on anterior and posterior abdominal wall. These features disappear after aspiration of the abscess. [22]

Differential Diagnosis [19]

1. Right subphrenic abscess.

   Recent abdominal surgery, inflammatory or perforating conditions of the intra abdominal viscera or abdominal trauma may suggest the diagnosis of subphrenic abscess; a lung and liver scan may help in differentiating it from the liver abscess.

2. Malignancy of liver.

   This may be a primary liver malignancy or secondary metastatic deposits in the liver. A hard nodular and markedly enlarged liver both with and without jaundice suggests malignancy.

3. Acute cholecystitis.

   Sudden onset of severe pain in right upper quadrant, nausea, vomiting fever and minimal icterus should suggest the diagnosis of acute cholecystitis. Murphy's sign will be positive.

4. Hydatid cyst of liver.

   Patients with hydatid cyst usually present with a mass in the upper abdomen. Cardinal features of liver abscess like fever, pain and tenderness are absent. Hydatid thrill can be elicited.

5. Alcoholic hepatitis.

   This condition occurs in chronic alcoholics. Jaundice is more frequent in alcoholic hepatitis.

6. Viral Hepatitis.

   Although liver abscess and viral hepatitis are both common in under developed countries, differentiating the two is not difficult. Complete loss of appetite, nausea, vomiting and absence of pain all favour the viral hepatitis.

7. Congestive liver of congestive cardiac failure or constrictive pericarditis.

   Oedema, breathlessness and hepatomegaly occurring occasionally in liver abscess may be mistaken for congestive cardiac failure. Absence of fever, severe pain, diffuse hepatomegaly and presence of engorged neck veins along with signs in the chest suggest involvement of heart.

8. Acute Abdomen

   Occasionally "Reflex ileus" in association with persistent vomiting, constipation and distension provides a picture which is difficult to distinguish from an acute intestinal obstruction.

Investigations

Haematological investigations:

Almost all patients with liver abscess have a haemoglobin level <12g% and a haematocrit level < 36%. There will be associated leukocytosis >10,000 cells/mm3, predominantly neutrophilic with absence of eosinophilia. [23]

Liver function tests

Elevation of bilirubin > 2 g/dl is common with pyogenic liver abscess. In an acute liver abscess, serum alkaline phosphatase will be normal and alanine transferase will be elevated and a reverse occurs in a chronic liver abscess. Hypoalbuminemia (Albumin <3g/dl) is present in about 90 % of the patients. [14]

Stool examination

The role of wet mount stool examination is limited as less than 30-40% of patients with amoebic liver abscess have concomitant intestinal amoebiasis. Microscopically similar but non-pathogenic Entamoeba dispar contributes for the high false positive rate. [24]

Examination of the stool is done by a combination of wet mount, iodine-stained concentrates, and trichrome-stained preparations. Haematophagous trophozoites of E histolytica require at least 3 fresh specimens as the trophozoites are very sensitive and may be excreted intermittently. Cysts must be differentiated morphologically from nonpathogenic Entamoeba hartmanni, Entamoeba coli, and Endolimax nana. Nonpathogenic E dispar cannot be differentiated morphologically and require fecal antigen detection. [9]

ELISA targeting the galactose inhibitable adherence protein of E. histolytica helps to differentiate between the two amoebae. Stool antigen detection facilitates early diagnosis before an antibody response. The drawbacks are the requirement for fresh, unpreserved stool specimens and the lack of intestinal amoebiasis in as many as 60% of patients with amoebic liver abscess. [13]

The PCR stool test is highly sensitive for detection of E histolytica and for distinguishing nonpathogenic amoebas. However, this test is expensive and less sensitive than ELISA for stool antigen. Real-time (rapid) PCR is being developed and is a sensitive test for detection of E histolytica. [25]

Serology

Indirect haemagglutination tests targeting the antibodies against E histolytica were used in the past. These tests remain positive after infection for many years and interpretation is difficult in patients from endemic areas.

These tests have been replaced by enzyme immunoassay (EIA) which is rapid and inexpensive. The EIA test detects antibodies specific for E histolytica in approximately. [26]

- 95% of patients with extraintestinal amoebiasis,
- 70% of patients with active intestinal infection, and

- 10% of persons who are asymptomatic cyst passers.

The EIA serology reverts to negative in 6 – 12 months following eradication of infection. Even in highly endemic areas, fewer than 10% of patients who are asymptomatic have positive amoebic serology findings.[26]

Initial test results may be negative in as many as 10% of patients with amoebic liver abscess, but repeat serology testing within a week will usually be positive.

Entamoeba histolytica galactose lectin antigen is detectable by Enzyme-linked immunosorbent assay (ELISA) in 75% of patients with amoebic liver abscess [26]. Initial antigen seropositivity is 96% with a reversal rate of 82% after 1 week of treatment with metronidazole. This test is useful for patients who present acutely, before an antibody response occurs. The serum sample is to be obtained before initiation of treatment, as medical therapy leads to rapid antigen loss. This test can be used for rapid diagnosis in highly endemic areas, where serology can be misleading.

Rapid testing for antigen and antibodies are being currently developed and are showing a promising future.[26]

Blood culture

Blood culture reveals growth in only about 50 % of cases. But there has been a poor correlation between the organism isolated from the abscess itself and the blood culture due to polymicrobial nature of the disease.

Diagnostic Aspiration

Under CT scan or ultrasound guidance, needle aspiration of cavity material can be performed.

Needle aspiration enables rapid recovery of material for microbiologic and pathologic evaluation. When the diagnosis is in doubt, it helps to identify whether the abscess is amoebic or pyogenic. [26]

Needle aspiration of the abscess can be performed with the initial diagnostic procedure.

Radiological investigations

Roentgenography

Chest X ray is abnormal in about 50% of cases. [19] Features include

1. Elevated right hemi diaphragm.
2. Pleural effusion.
3. Atelectasis.
4. Pneumonitis.

Abdominal X ray may reveal

1. Hepatomegaly.
2. Gas within the abscess cavity due to
    a. Infection by gas forming organisms.
    b. Secondary infection of amoebic abscess.
    c. Prior percutaneous intervention.
    d. Rupture into a hollow viscous.
3. Air in the portal vein if associated pylephlebitis is present. [14]
4. Aerobilia may be present if cholangitis is present. [27]

Absence of the findings however doesn't rule out liver abscess.

Ultrasonography

Ease of access and low cost has made ultrasonography a boon in evaluation and treatment of live abscess. USG can identify lesions as small as 2cm. The typical lesion is a hypo echoic round lesion with an echogenic wall, acoustic enhancement and internal echoes. Ultrasonography allows for close evaluation of the biliary tree and simultaneous aspiration of the cavity. The major benefits of this technique are its portability and diagnostic utility in patients who are too critical to undergo prolonged radiologic evaluation or to be moved out of monitored setting. [14]

The drawbacks of USG include. [19]

1. Doesn't visualize the dome of liver.
2. Multiple micro abscesses may not be seen.
3. Fatty infiltration increase echogenicity of liver and decrease the sensitivity of the study.
4. Operator dependence affects its overall sensitivity.

CT scan [14]

Computed tomography scanning has the advantage of being able to detect intrahepatic lesions as small as 0.5 cm and also image the entire abdomen.

A classical lesion of liver abscess will be a hypo dense cystic lesion with thick segmental wall enhancement and surrounding low density oedema. A central large lesion can be surrounded by adjacent "daughter" abscess representing coalescing of multiple small abscess – Cluster sign, is suggestive of bacterial cause. [28]

MRI

Magnetic resonance imaging can better characterize hepatic lesions compared to CT. MRI can distinguish liver abscess from other cystic and necrotic lesions. [9] High cost, length of study and lack of easy access for drainage procedure has limited the usefulness in management of liver abscess. [11]

Other imaging

Technetium – 99m liver scanning is useful test for differentiating an amoebic liver abscess from a pyogenic abscess; however, it is not a first-line test. [26]

Amoebic liver abscesses do not contain leukocytes; they appear as cold lesions on hepatic nuclear scanning, with a typical hot halo or a rim of radioactivity surrounding the abscess. [26]

In contrast, pyogenic liver abscesses contain leukocytes and, therefore, typically appear as hot lesions on nuclear scanning.

Gallium scanning is helpful in differentiating pyogenic abscess but requires delayed images, which makes the test less helpful. Gallium and Technetium 99m scans have largely been abandoned in their role of investigating and treatment of liver abscess due to advances in CT and USG. [9]

Treatment

Pyogenic liver abscess

An untreated hepatic abscess is nearly uniformly fatal due to the complications that follow. Management of pyogenic liver abscess involves appropriate antibiotic therapy and drainage of abscess, followed by identifying the source of abscess and successful treatment of the same.

Appropriate antibiotic therapy involves the identification of the organism by blood culture or culture of the pus from the abscess. Treatment is initiated with broad spectrum antibiotics covering gram positive and negative organisms and anaerobic bacteria. A prolonged course of antibiotics lasting 4 – 6 weeks is required as there is a perception of presence of avascularity in the abscess. [14]

The differences in clinical presentation of pyogenic and amoebic liver abscess are given in the table below. [19]

Clinical features	**Amoebic liver abscess**	**Pyogenic liver abscess**
Age (years)	20 – 40	>50
Male : Female	>10:1	1.5:1
Solitary vs. Multiple	Solitary 80%	Solitary > 50 %
Location	Usually Right lobe	Usually right lobe
Travel to endemic area	Yes	No
Diabetes and other forms of Immunosuppression	No	Yes
Alcohol use	Common	Common
Jaundice	Uncommon	Common
Elevated ALP	Common	Common
Positive blood culture	No	Yes
Positive amoebic serology	Yes	No

Drainage procedures

Most liver abscesses require some form of drainage, whether it is closed aspiration, percutaneous drainage or surgical.

Closed aspiration

Giorgio and colleagues reported a series of 115 patients who underwent percutaneous aspiration and reported a 98.3 % success rate with no mortality or morbidity. [29] Percutaneous aspiration is usually undertaken under USG or CT guidance. At present, the indications for closed needle aspiration include [14]:

1. Lack of improvement with subsidence of symptom and signs in 48-72 hours after medical line of treatment.
2. Abscess greater than 5 cm in diameter. [19].

Percutaneous Aspiration is not recommended in patients with [14]

1. Multiple large abscesses.
2. A known intra- abdominal source that requires surgery.
3. An abscess of unknown aetiology.
4. Abscess that would require transpleural drainage.
5. Ascites.

Complications

1. Haemorrhage: External bleeding from punctured site is usually of short duration and is only about 5 to 10ml. Inter or intraperitoneal haemorrhage can occur from perforation of portal vein, hepatic veins and aberrant or intercostal arteries. Intrahepatic haematoma can occur but usually is asymptomatic. Rupture of intrahepatic haematoma into a bile duct may lead to haemobilia.
2. Puncture of gall bladder is usually followed by biliary peritonitis and surgical intervention may be warranted.
3. Pleurisy, perihepatitis and pneumothorax can occur. [29]
4. Death due to vagal shock can occur very rarely.

Percutaneous aspiration is less invasive, less expensive; however recurrence rates and the requirement for surgical intervention may be greater who undergo aspiration alone.

Catheter drainage

In 1998, a randomised control trial conducted by Rajak et al compared percutaneous aspiration to catheter drainage. [30] They reported a 60% success with percutaneous aspiration versus a 100% success with catheter drainage. The indications and complications are similar to percutaneous aspiration.

Advantages

1. It can be used as therapeutic and diagnostic procedure.
2. Relatively safe economic and informative.
3. Follow up of the resolution of the abscess cavity can be ascertained by studies like air cavitogram or by use of dyes. [31]

Disadvantages

1. Catheter blockade can occur and require re – insertion.
2. Can't be used when there is evidence of threatening rupture.

Open surgical drainage

Surgical drainage had been an accepted treatment modality since 1938. [1] Initially abscesses were drained extraperitoneally to avoid contamination of the peritoneum. [9] With the advent of antibiotics and improvements in post operative care, transperitoneal exploration is a safe surgical approach. The transperitoneal approach has the advantage of the ability to [14]:

1. Treat the inciting pathology in the remainder of abdomen or pelvis.
2. Gain access and exposure of entire liver for evaluation and treatment.
3. Access the biliary tree for cholangiography and bile duct exploration.

Surgical drainage is currently reserved for patients that [11]

1. Ruptured liver abscess.
2. Have failed non – operative therapy.
3. Those needing surgical treatment of the underlying source.
4. Multiple macro abscesses.
5. Concomitant ascites.

For high posterior lesions, a posterior transpleural approach is advocated. This allows easier access to the abscess, but the advantage to identify multiple lesions or a concurrent intra-abdominal pathology is lost.

Laparoscopic surgical drainage [32, 33]

Laparoscopic drainage is an attractive alternative for patients requiring open surgical drainage. Laparoscopic surgery has the advantages of both open surgery and the minimal invasiveness of percutaneous drainage. The advantages of laparoscopic surgery in terms of reduced analgesia requirements, reduced morbidity, faster postoperative recovery and shorter hospital stay compared to laparotomy are well documented. Laparoscopic localization of liver abscess may be more difficult than at open surgery due to lack of tactile feedback. However, aspiration with a long endoscopic or spinal needle may aid localization. Laparoscopic ultrasonography is useful in this respect.

There is no single best treatment modality for pyogenic liver abscess. Therapy has to be individualised to each patients to achieve the best possible outcome.

Factors associated with poor outcome in Pyogenic liver abscess [14]:

- Age >70 years.
- Diabetes mellitus.
- Associated malignancy.
- Biliary aetiology.
- Multiple abscesses.
- Septicaemia.
- Polymicrobial bacteraemia.
- WBC count >20,000 cells/mm3.
- Increasing bilirubin.
- Increasing SGOT.
- Albumin <2 g/dL.
- Aerobic Abscess.
- Significant complication like rupture and peritonitis.

Follow up

Patients will require prolonged parenteral antimicrobial therapy that may continue after discharge. Radiologic evaluation to document progress of therapy after discharge is required.

Amoebic liver abscess

Majority of the amoebic abscess can be treated with medical management. Drainage procedures, regardless of the approach, are reserved for patients with questionable diagnosis or when complications ensue.

Anti amoebic drugs

Classification [34]

1) Tissue amoebicides.
    a) Intestinal and extraintestinal amoebiasis:
        i) Nitroimidazoles: Metronidazole, Tinidazole, Secnidazole, Ornidazole, Satranidazole.
        ii) Alkaloids: emetine, Dehydroemetine.
    b) For extraintestinal amoebiasis only: Chloroquine.
2) Luminal amoebicides:
    a) Amide: Diloxanide furoate.
    b) 8 – Hydroxyquinolones: Quinidochlor, Iodoquinol.
    c) Antibiotics: Tetracyclines.

Metronidazole: Prototype Nitroimidazole compound. It has broad spectrum cidal activity against protozoa and anaerobic bacteria. Achieves high concentration in liver with small amounts of drug, absorbs rapidly, rapidly excreted without cumulative effect. The current recommendation for liver abscess is 750 mg TDS for 10 days. In serious cases of liver abscess 1g may be infused IV followed by 0.5g 12^{th} hourly till oral therapy is instituted. For mild intestinal disease 400mg TDS for 7 days is recommended. Adverse effects include Headache (10%), dizziness, nausea, anorexia, Heavy coating of tongue, brownish urine, metallic taste, ataxia(<1%), seizures, paraesthesias, disulfiram like reaction with alcohol.[34]

Tinidazole: A congener of Metronidazole with slower metabolism resulting in less frequent dosing. 2g OD for 3 days or 0.6g BD for 5 – 10 days. The incidence of adverse effects is lower.

Chloroquine: useful in hepatic amoebiasis only as it is completely absorbed in the upper intestine without achieving concentration in the intestinal wall. Amoebae do not develop resistance to chloroquine but duration of treatment is longer and relapses are more frequent. Dose for amoebic liver abscess is 600mg base for 2 days followed by 300mg base for next 2 – 3 weeks. It is contraindicated in patients with retinopathy and psoriasis.

Other adverse effects include G.I. disturbances, headache, visual disturbances and Pruritis.

Diloxanide furoate: is a highly effective luminal amoebicide which directly kills the trophozoites responsible for cyst production. Effective in cyst passers and therefore used for treatment of asymptomatic carriers, family members and intimate contacts of patients diagnosed with liver abscess. Dosage is 500 mg TDS for 10 days. Adverse effects include flatulence, nausea, vomiting, pruritis and urticaria.

Therapeutic protocol [9]

Metronidazole is the current drug of choice. It is administered as a single drug (750mg TDS) after diagnosis, with concomitant correction of hypoprothrombinemia, hypoproteinemia and anemia. If improvement occurs within 48 – 72 hours, metronidazole is continued till 14 days. A luminal agent such as Diloxanide furoate (500 mg PO, TDS. x 10 days) must be administered following metronidazole therapy for the eradication of intestinal infection as a part of the complete treatment. In patients who do not respond, Emetine or Dehydroemetine is added.

Freeman et al in their study of 36 patients with amoebic liver abscess showed percutaneous needle aspiration is safe, enhances clinical recovery,

and accelerates resolution particularly in patients with large abscess cavities. [35]

Therapeutic aspiration of amoebic liver abscess is considered when there are [36]:

1. High risk of abscess rupture, as defined by cavity size greater than 5 cm;
2. Left lobe liver abscess, which is associated with higher mortality and frequency of peritoneal leak or rupture into the pericardium;
3. Failure to observe a clinical response to medical therapy within 5-7 days; and
4. A difficulty in differentiating from a pyogenic liver abscess.
5. Age older than 55 years.

In endemic areas as many as 50% of patients may require aspiration, as many present late and have multiple abscesses.

Routine needle aspiration offers only minimal benefit over medical care alone. Needle aspiration is avoided for uncomplicated amoebic liver abscess unless one of the above indications exists. Prompt medical care decreases the need for aspiration.

Catheter drainage is considered when large volume of abscess needs to be drained. Rajak et al demonstrated that catheter drainage may be better

compared to needle aspiration, but the average time for clinical improvement, mean hospital stay were similar in the two groups.[30] Generally, surgical drainage is not necessary and should be avoided; however, open surgical drainage is considered when the abscess is inaccessible to needle drainage or a response to therapy has not occurred.[14]

Evidence of pulmonary, peritoneal or pericardial extension is a dreaded complication and an indication for drainage of the liver abscess with an intercostal tube or catheter into a closed-circuit collection system. Failure to adequately control the abscess by these means or when there are increasing signs or features of peritonitis or fistulisation into a hollow viscus or secondary infection with septicaemia constitutes an indication for laparotomy. [9, 14, 19]

Factors associated with poor outcome in Amoebic liver abscess. [14].

- Serum bilirubin >3.5 mg/dL.
- Encephalopathy.
- Albumin <2 g/dL.
- Multiple abscess cavities.

Follow up

After clinical cure, patients are usually asymptomatic and ultrasonographic follow up demonstrates evidence of persistent hypoechoic lesion. The mean time for the disappearance of the sonographic abnormality is 6 - 9 months. The patterns of resolution seen on sonographic follow up include[37]:

Type I: where complete disappearance of the cavity occurs within 3 months,

Type II: a rapid reduction till 25% of the original cavity size and then a delayed resolution. Clinical resolution does not correlate with radiologic resolution; the result of the therapeutic intervention is monitored by clinical criteria rather than USG.

Fungal liver abscess.

It is an unusual cause of liver abscess whose recent incidence is on the rise.

Aetiology.

The common causative organisms causing liver abscess include:

- Candida species like C. albicans, C. glabrata.
- Cryptococcus sp.
- Asperigillus sp.

Predisposing factors.

Fungal liver abscess is more common in patients in

- Immunocompromised states.
- Diabetes mellitus.
- Hematologic malignancies like lymphoma and leukemia.

Few cases are reported in immune competent patients also. [38]

Investigations

Ultrasonography and CT reveal multiple abscesses which are suggestive of fungal nature in the predisposed patients.

Fungal culture and microscopy of the aspirate are diagnostic.

Treatment

Amphotericin B and Fluconazole are the drugs of choice.

Percutaneous aspiration can be done in cases where conservative management fails.

Open surgical drainage is reserved for resistant cases.

FDG PET helps in the evaluation of treatment response in fungal liver abscesses. [39]

Tubercular Liver abscess.

Tubercular liver abscesses are extremely rare. It was first described by Bestowe in 1858.

Etiology

Tubercular liver abscess is most commonly due to haematogeneous spread from co existing pulmonary or gastro intestinal tuberculosis. Formation of tubercular liver abscess in patients with miliary tuberculosis is also explained. [40]

Clinical features.

The clinical features are non specific which include fever, abdominal pain, nausea and anorexia.

Jaundice is rarely seen.

Hepatomegaly with smooth enlargement and right hypochondriac tenderness are the most common signs. [41]

Investigations

USG and CT scan reveal either granulomatous tubercle, calcification depending on the activity of the disease. 'Honey comb' pattern of septations or 'cluster sign' may be evident on CT scan. [42, 43]

Confirmation of the diagnosis is made by demonstrating AFB in the pus aspirate by microscopy or culture.

PCR is a useful investigation in diagnosis of tubercular liver abscess and helps to differentiate M. tuberculosis from other mycobacterium.

Treatment

Antitubercular therapy using Isoniazid, Rifampicin, Pyrizinamide and Ethambutol for a period of 1 year can be considered in all patients. Percutaneous aspiration is a useful adjunct to antitubercular therapy. Prognosis is generally good for isolated tubercular liver abscess.

Objectives

1. To study the etiology, clinical manifestations of liver abscesses.
2. To know the clinical response in patients with liver abscess, undergoing open surgical drainage and percutaneous needle aspiration.

Materials and Methods

Patients admitted at Chengalpattu Medical College and Government hospital during October 2010 to October 2012 and diagnosed with liver abscess were included into the study.

Inclusion criteria.

1. Patients aged between 18 and 65 years admitted with

    a. A diagnosis of liver abscess with USG/ CT with size > 5cm in diameter.

Exclusion criteria.

1. Patients aged above 65 and below 18 years.
2. Patients with liver abscess with size <5cm in diameter.
3. Patients with liver abscess ruptured into peritoneal, pleural or pericardial cavity.
4. Patients with liver abscess and coexisting ascites.
5. Patients who do not consent for the study.

Patient data collection

A detailed history was collected from the patients who were selected for the study. The investigations were done and recorded as in the proforma. All patients received intravenous Ciprofloxacin 200 mg bd and

Metronidazole 800 mg tds received as the initial treatment. Patients were divided into two groups and consent was taken regarding the procedure being undertaken.

Group I underwent USG guided percutaneous needle aspiration.

Group II received open surgical drainage.

Patients' coagulation profile was normalised before any intervention.

Percutaneous needle aspiration.

- The patient was kept NPO for six hours.
- 0.6 mg Atropine was injected to counteract vagal attack.
- Aspiration was undertaken in an operation theatre with precautions for asepsis.
- The site and depth of abscess was assessed and marked using USG guidance prior to the procedure.
- The patient was put in semi-recumbent position, leaning against a backrest.
- The selected area for aspiration was infiltrated with 5% Xylocaine after surgical draping.
- Percutaneous aspiration was done with 18 gauge large bore needle. Aspiration was done using 10 ml syringe and continued until no more pus could be evacuated or until patient became distressed.

- The aspiration site was sealed with tincture Benzoin. An adhesive plaster covering the right lower chest was applied.
- The aspirated pus was sent for culture and sensitivity or microscopy accordingly.
- Adequate analgesia was provided for the patient.
- Patient was observed for twenty four hours with monitoring of the temperature, pulse, respiratory rate and abdominal girth to identify any signs of haemorrhage or peritonitis.
- Patients were started orally the next day as tolerated.

Open surgical drainage.

- Patients were made to fast for at least six hours and abscess localised by USG and marked.
- General or epidural anaesthesia was administered.
- With patient in supine position and meticulous surgical toileting of the abdomen was done. The abdomen was opened with a midline vertical incision.
- The liver was isolated from the rest of the peritoneal cavity by packing surgical mops. The abscess site was confirmed by needle aspiration.

- A hepatotomy was made with an electrocautery to open the abscess cavity. The abscess is completely drained out with suction. A thorough wash was given with saline.
- Care was taken to not break inter - running biliary radicles within the abscess cavity. A thorough examination of the intra – abdominal contents was made to rule out other intra – abdominal pathology.
- Large bore tube drain was placed and brought out through a separate stab incision.
- Abdomen was closed in layers after confirming the instrument and mopp count.
- Patient was monitored regularly with watch on the temperature, pulse, respiration in the post operative period.
- Oral feeds were started as tolerated by the patient. Drains were removed accordingly after confirming that there was no bile leak.

Post procedure follow up

Patients were examined daily for clinical improvement. Improvement of pain, fever, anorexia, and hepatomegaly were considered as a criterion for successful treatment. Complications arising from either of the procedures were noted and appropriate measures were undertaken.

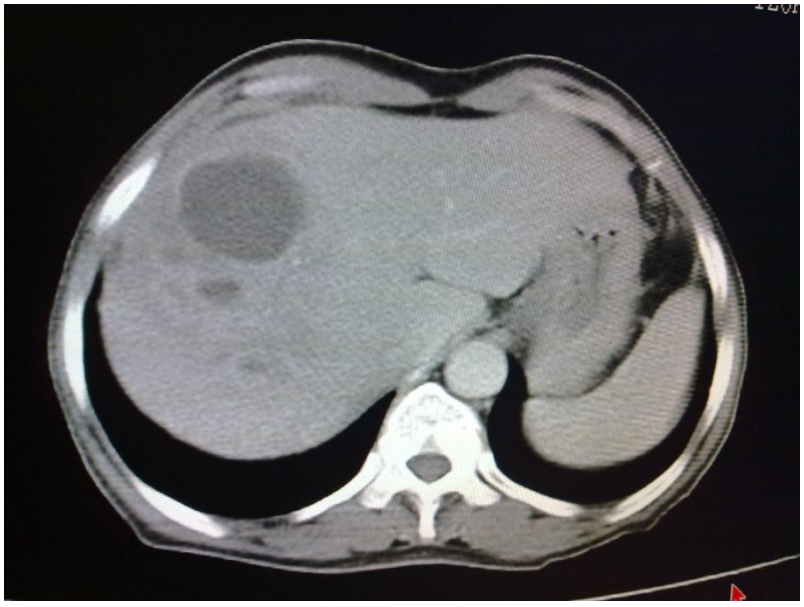
Ultrasonography was done as indicated. Relapses were noted and repeat aspirations were performed when necessary.

Cure was defined as improvement clinically with subsidence of fever, and local signs, symptoms, decrease in WBC count and follow-up ultrasonography showed reduction in size < 3 cm in diameter and no evidence of relapses. Mean Hospital Stay was recorded.

Patients were followed up for a minimum period of 3 months for recurrent attacks or development of complications and to monitor the efficacy of the treatment given.

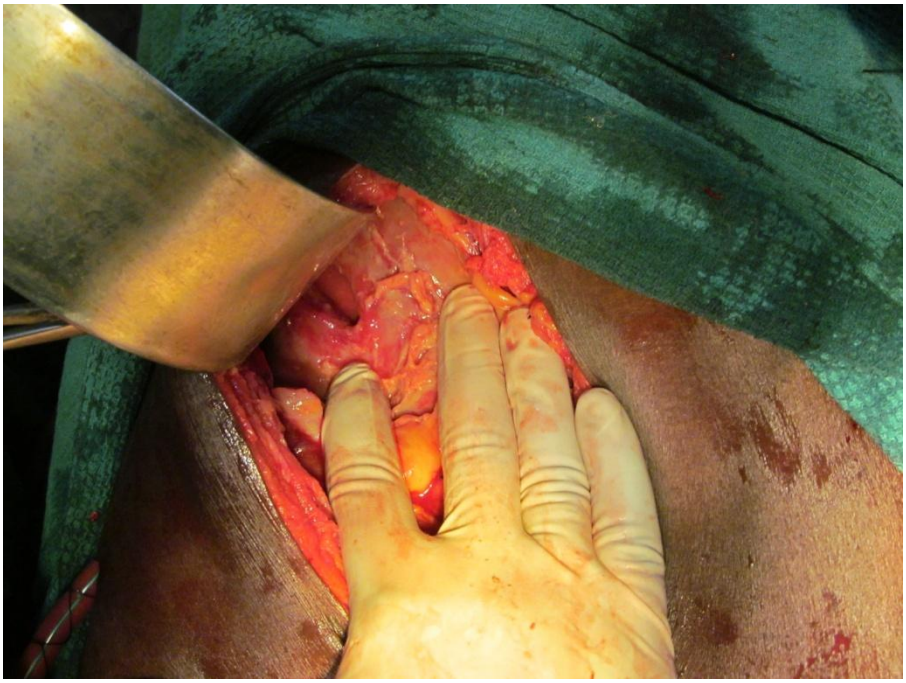
PHOTOGRAPHS

X ray showing an Air – fluid level in the liver.



CT showing the "Cluster sign" in a Pyogenic Liver abscess.

Percutaneous Aspiration of Pyogenic Liver Abscess being undertaken.



Intraoperative Photograph of Liver Abscess showing the Abscess cavity.

Observations and Results

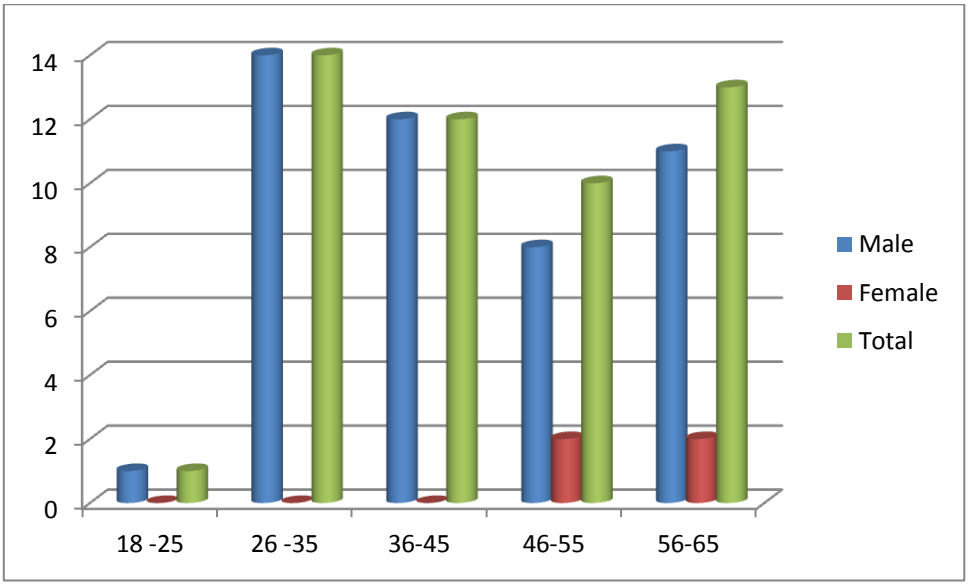
Patient data was collected using the proforma enclosed in the Annexure and tabulated using Microsoft Excel 2007.

Tables and graphs were generated using the Microsoft Excel 2007.

All statistical analysis was done using Winstats (v1.11) software. Standard deviation and Chi square test were used and ‘p’ value was determined.

Table 1: Table showing age and sex wise distribution of the studied population.

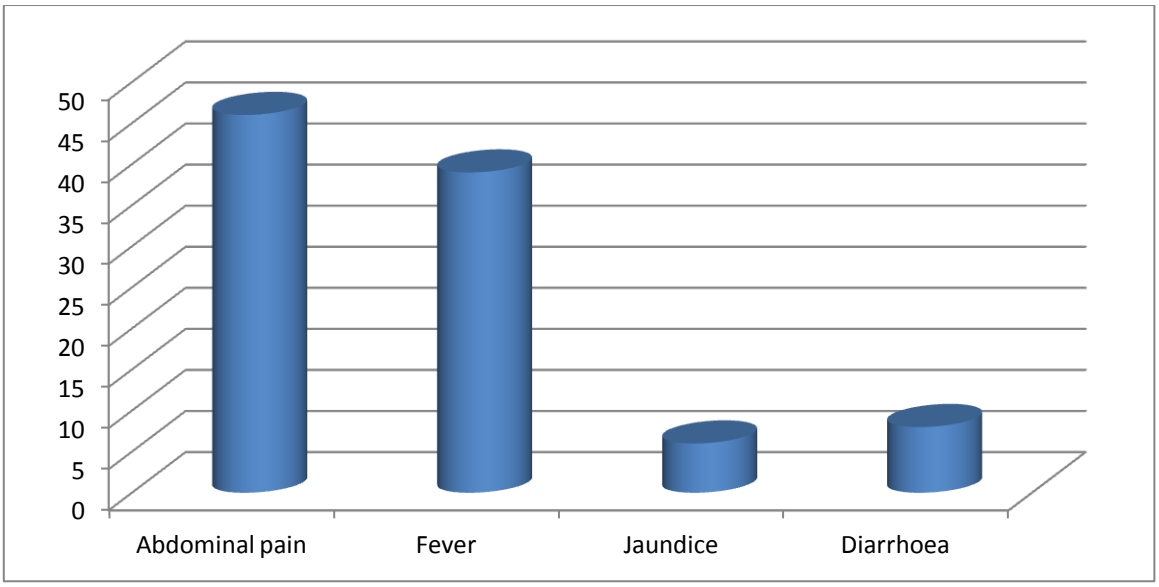
Age	Male	Female	Total	Percentage
18 -25	1	0	**1**	**2**
26 -35	14	0	**14**	**28**
36-45	12	0	**12**	**24**
46-55	8	2	**10**	**20**
56-65	11	2	**13**	**26**
Total	**46**	**4**	**50**	**100**



The studied population was between the age group of 18 to 65 years. The majority of the patients were in the age group of 26 - 35 years. The mean age of the patients was 44.8 years. Majority of the studied population were males (92 %) with females constituting 8 %.

Table 2: Table showing the symptoms associated with liver abscess in the studied population.

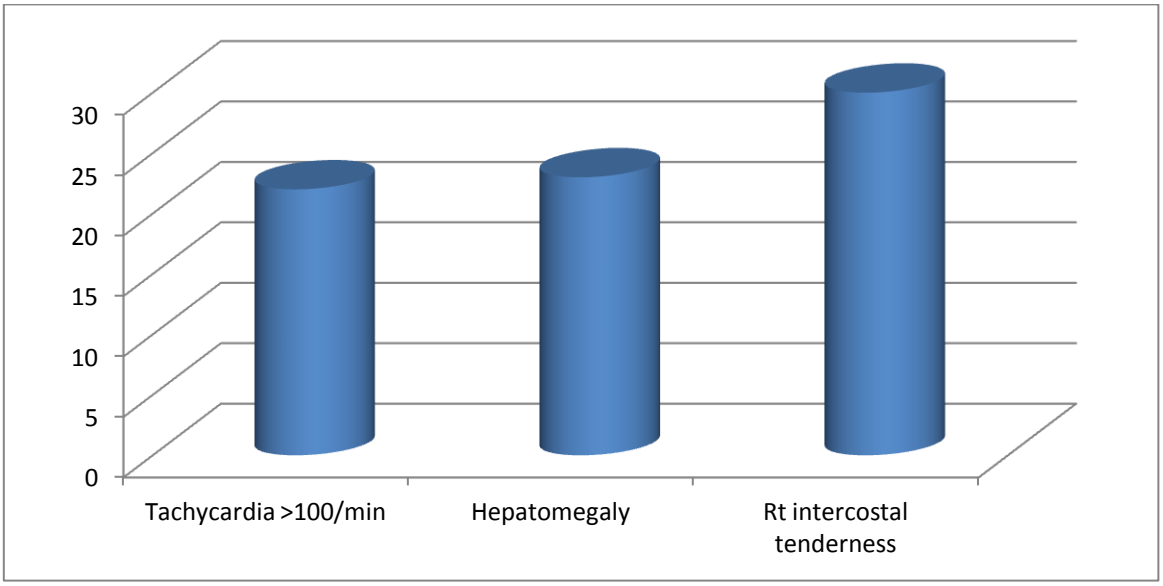
Symptom	No of patients	Percentage
Abdominal pain	46	92 %
Fever	39	78 %
Jaundice	6	12 %
Diarrhoea	8	16 %



Abdominal pain was the most common symptom (92 %) present in patients with liver abscess. Fever was present in 78 % of the patients.

Table 3: Table showing the most common signs associated in patients with liver abscess.

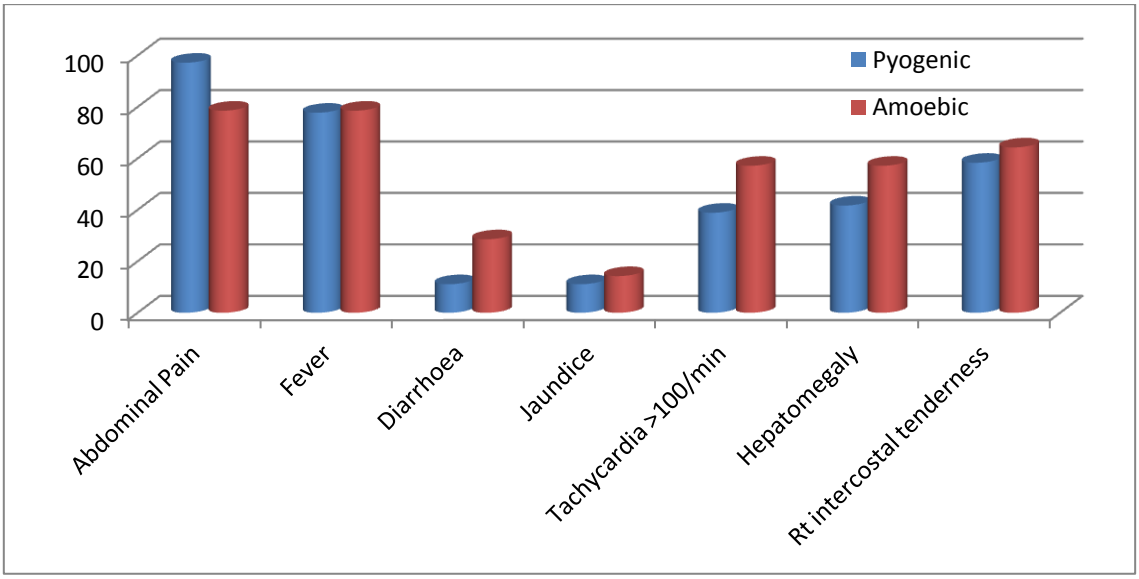
Signs	Number of patients	Percentage
Tachycardia >100/min	22	44
Hepatomegaly	23	46
Rt intercostal tenderness	30	60



Right lower intercostal tenderness was the most consistent sign and was present in 60 % of the patients while tachycardia (>100/min) was present in 44 % and hepatomegaly was present in 46 % of the patients.

Table 4: Table showing the signs and symptoms of liver abscess, according to its aetiology.

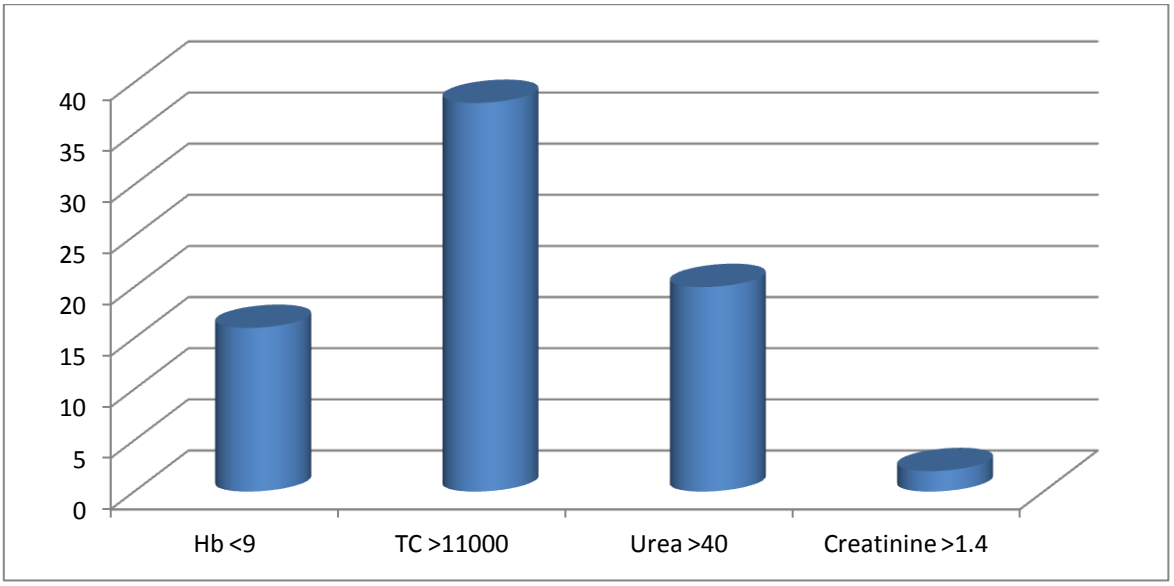
Signs and Symptoms	Pyogenic	Amoebic
Abdominal Pain	97.22 %	78.57 %
Fever	77.78 %	78.57 %
Diarrhoea	11.11 %	28.57 %
Jaundice	11.11 %	14.28 %
Tachycardia >100/min	38.89 %	57.14 %
Hepatomegaly	41.67 %	57.14 %
Rt intercostal tenderness	58.33 %	64.28 %



Abdominal pain was the most consistent complaint in both pyogenic and amoebic liver abscess. Right lower intercostal tenderness was the most consistent sign in both pyogenic and amoebic liver abscess.

Table 5: Table showing the number of patients with deranged investigations in the studied population.

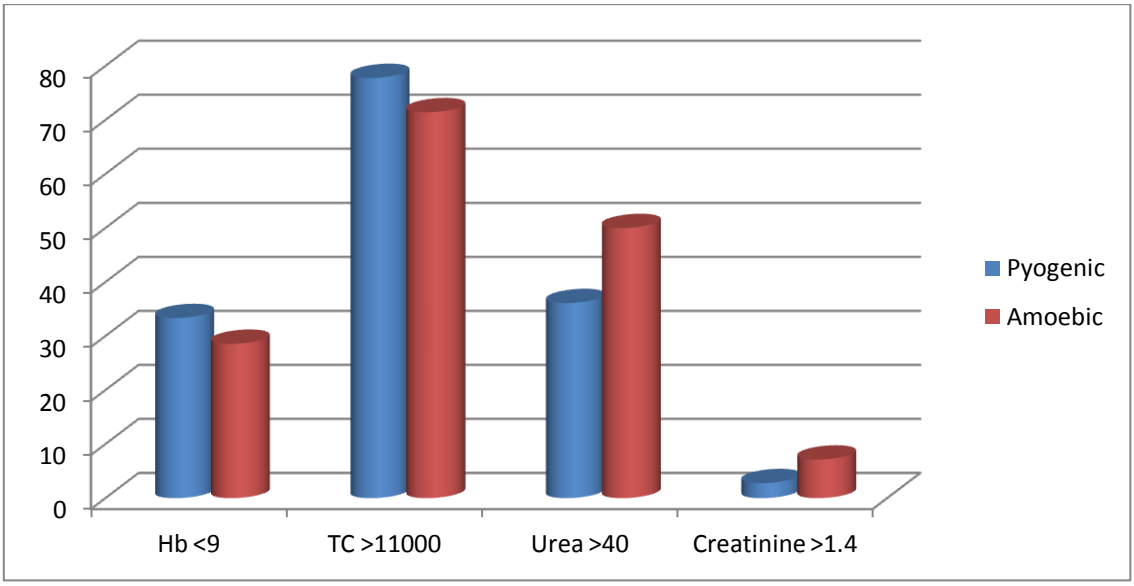
Investigations	Total	Percentage
Hb <9	16	32
TC >11000	38	76
Urea >40	20	40
Creatinine >1.4	2	4



76 % of the patients had a total count > 11,000. 40 % of patients had urea > 40mg/dl and 32 % had haemoglobin < 9 g/dl.

Table 6: Table showing the number of patients with deranged investigations in the studied population based on the type of liver abscess.

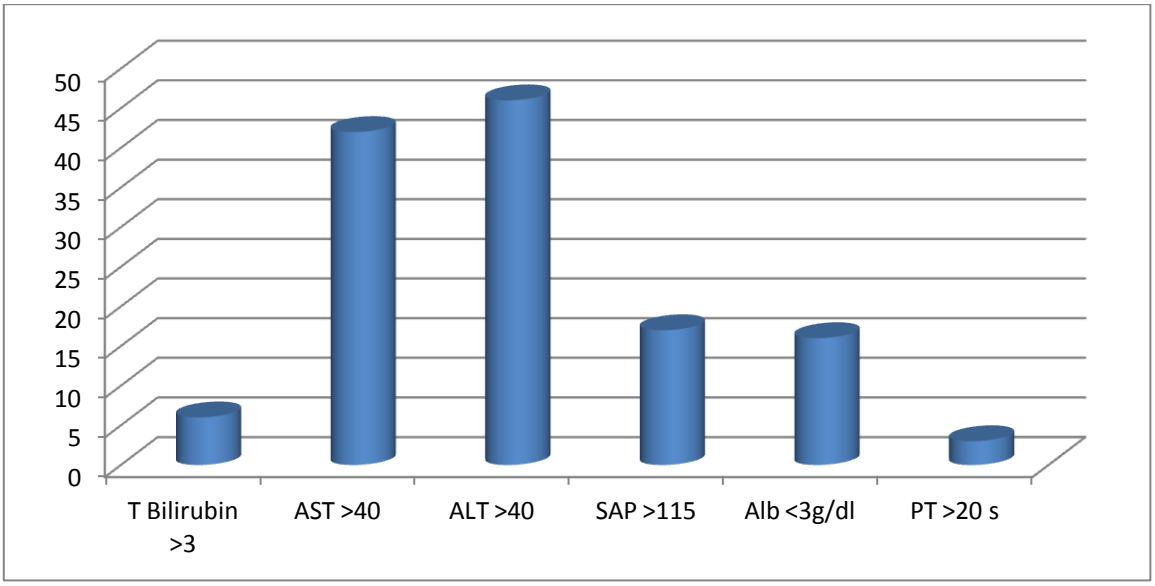
Investigations	Pyogenic	Amoebic
Hb <9	33.33 %	28.57 %
TC >11000	77.78 %	71.43 %
Urea >40	36.11 %	50 %
Creatinine >1.4	2.78 %	7.14 %



Total count > 11,000 cells/mm3 was seen in 77.8 % and 71.4 % of pyogenic and amoebic liver abscess respectively. Elevation of urea was more common in amoebic liver abscess compared to pyogenic liver abscess.

Table 7: Table showing the number of patients with abnormal liver function test parameters.

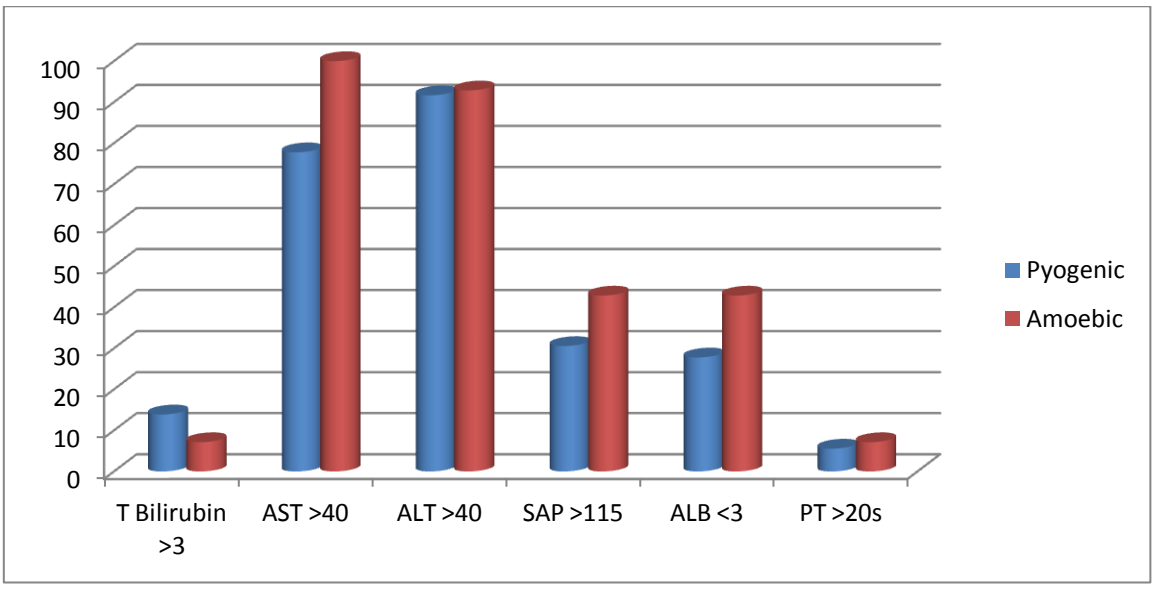
Investigations	Total	Percentage
T Bilirubin >3	6	12 %
AST >40	42	84 %
ALT >40	46	92 %
SAP >115	17	34 %
Albumin <3g/dl	16	32 %
PT >20 s	3	6 %



Most of the patients had elevation of AST and ALT levels. 32 % of patients had Albumin < 3 g/dl. 34 % of patients had elevation of Serum Alkaline phosphatase.

Table 8: Table showing the number of patients with abnormal liver function test parameters based on the type of liver abscess.

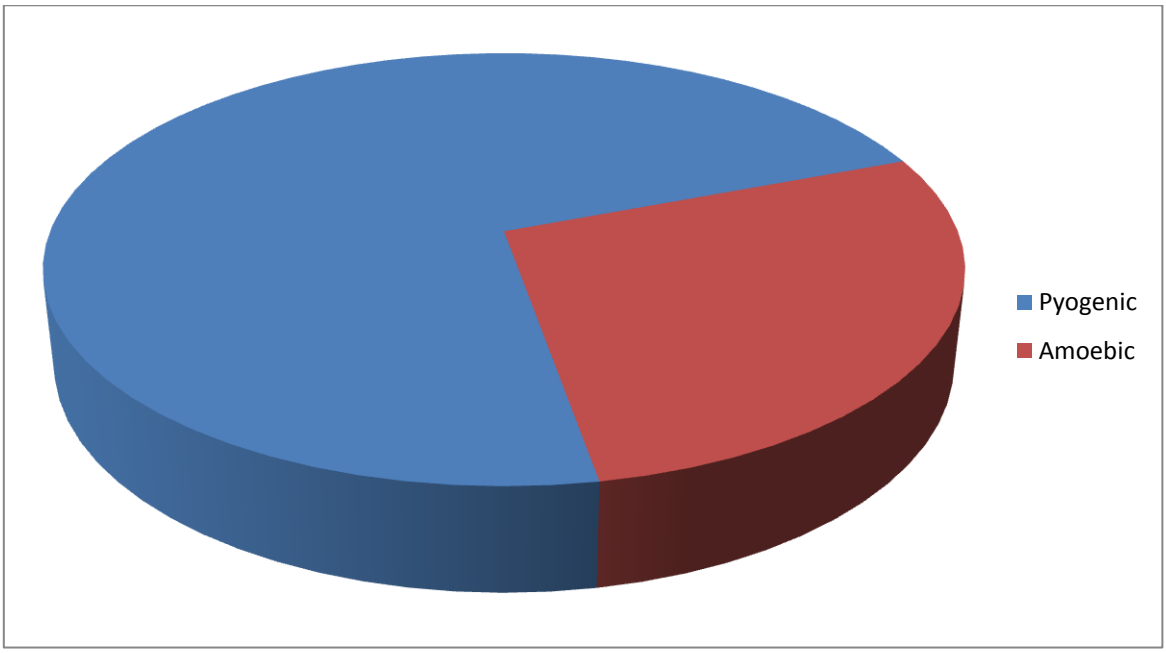
Investigations	Pyogenic	Amoebic
T Bilirubin >3	13.89 %	7.14 %
AST >40	77.78 %	100 %
ALT >40	91.67 %	92.86 %
SAP >115	30.56 %	42.86 %
ALB <3	27.78 %	42.86 %
PT >20s	5.56 %	7.14 %



All patients with amoebic abscess had elevation of AST. Elevation of AST and ALT were the most common parameter to be elevated in both amoebic and pyogenic liver abscesses.

Table 9: Table showing the distribution of patients based on the type of liver abscess.

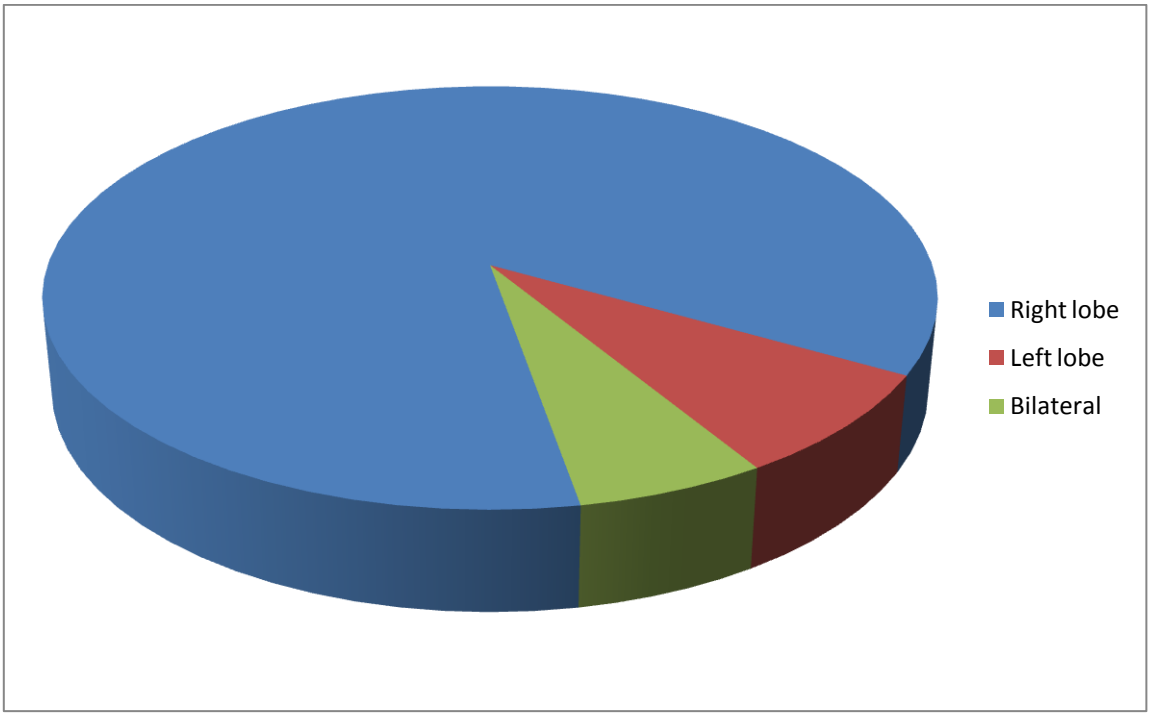
	Total	Percentage
Pyogenic	36	72
Amoebic	14	28



72 % of the studied population had pyogenic abscess while the remaining 28 % had amoebic abscess.

Table 10: Table showing the distribution of patients in the studied population based on the lobe of involvement.

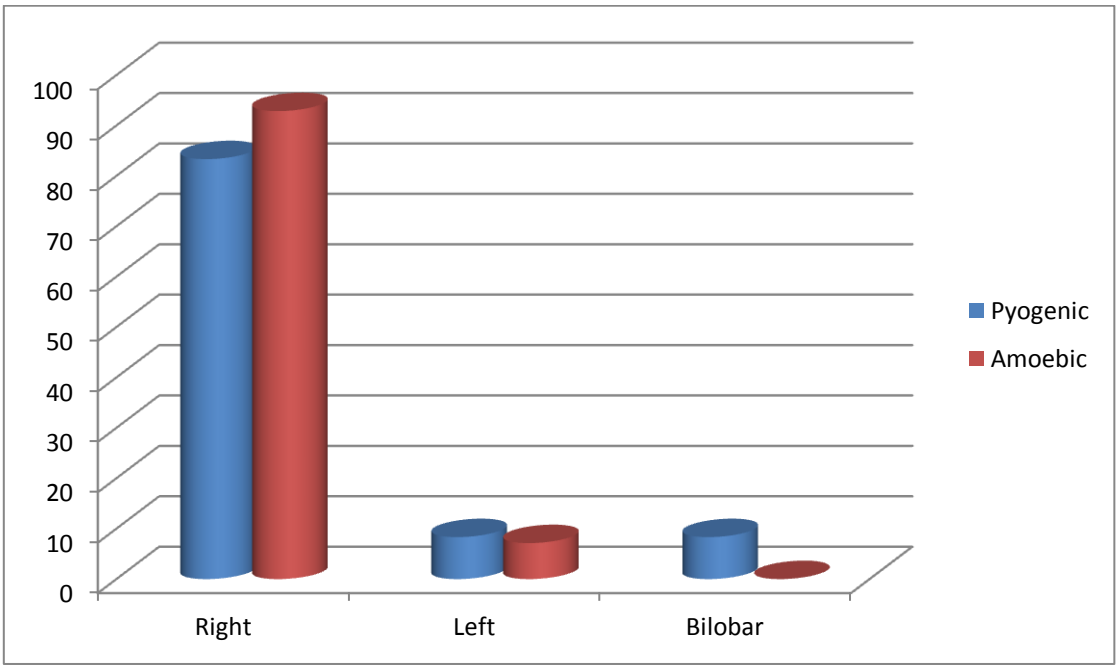
	No. of Patients	Percentage
Right lobe	43	**86**
Left lobe	4	**8**
Bilateral	3	**6**
Total	**50**	**100**



Involvement of the right lobe was seen predominantly in 86% of cases. Isolated left lobe involvement was seen in 8 % of cases and 6 % had involvement of both lobes of liver.

Table 11: Table showing the distribution of patients in the studied population based on the lobe of involvement and the type of abscess.

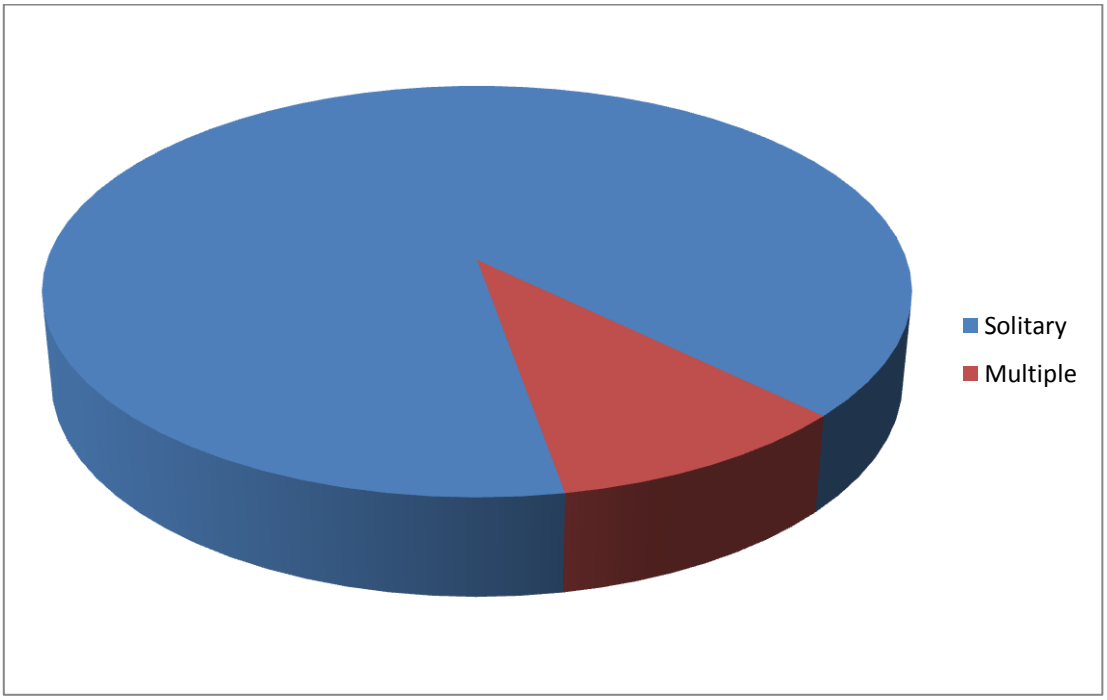
	Pyogenic	Amoebic
Right	83.33 %	92.86 %
Left	8.33 %	7.14 %
Bilobar	8.33 %	0



Predominant right lobe involvement was seen in both pyogenic and amoebic abscess. Simultaneous involvement of both lobes was seen in pyogenic liver abscess only.

Table 12: Table showing the occurrence of liver abscess as a solitary or multiple lesions.

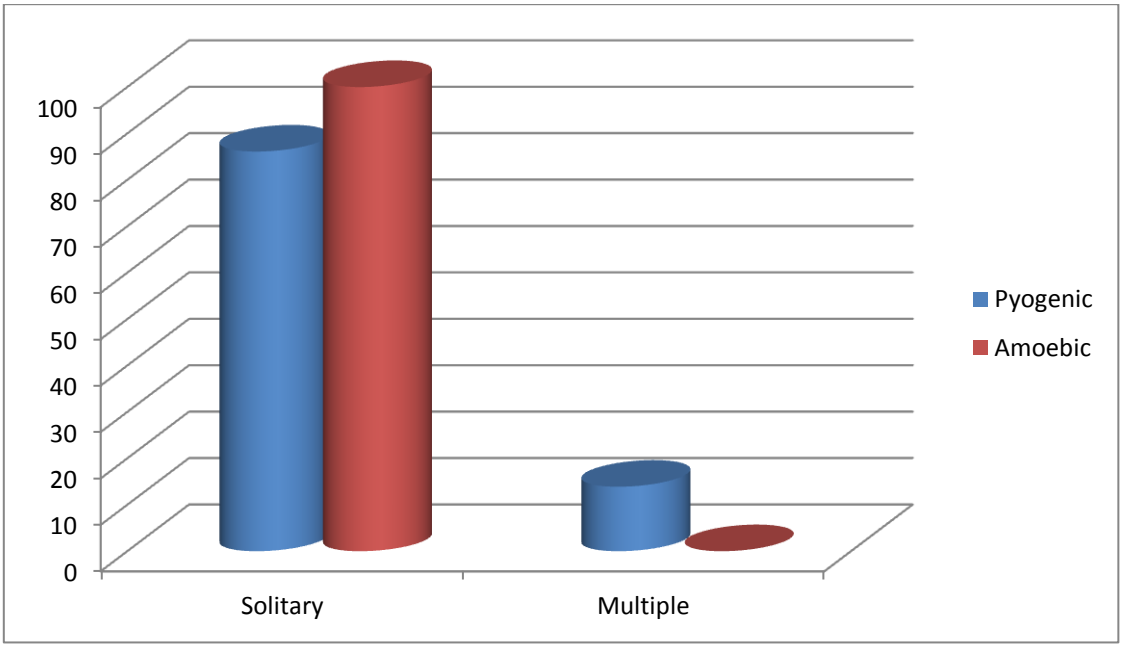
	Total	Percentage
Solitary	45	90
Multiple	5	10
Total	**50**	**100**



90% of the liver abscess occurred as a solitary lesion and the remaining 10 % had multiple lesions.

Table 13: Table showing the occurrence of liver abscess as solitary or multiple lesions based on the aetiology.

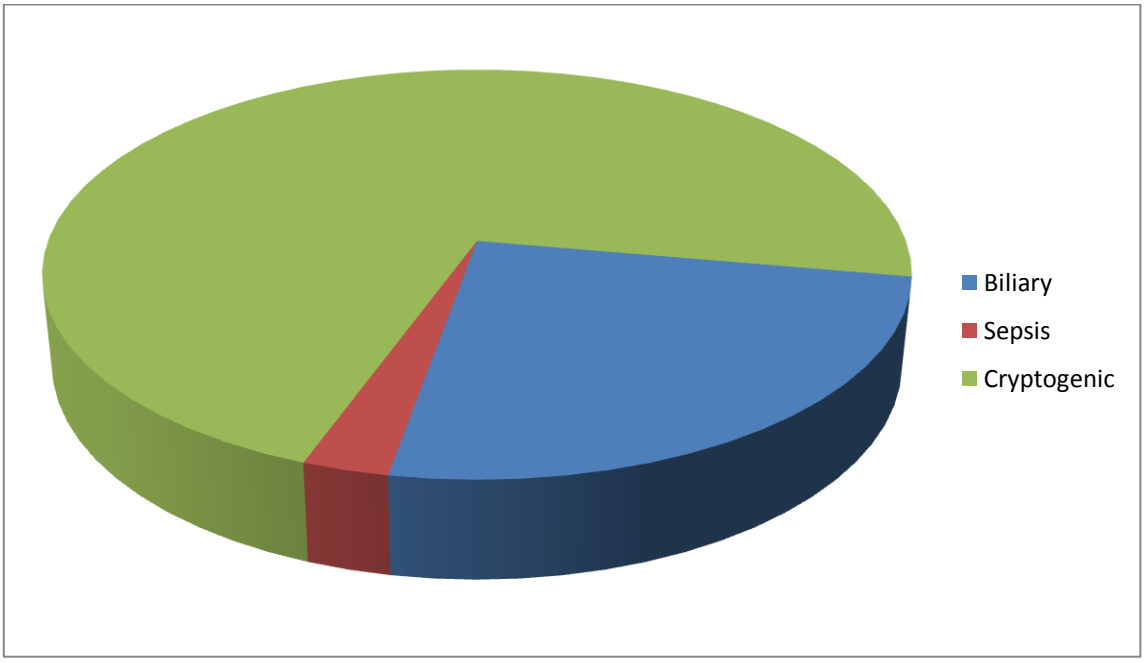
	Solitary	**Multiple**
Pyogenic	86.11 %	13.89 %
Amoebic	100 %	0



All of amoebic abscess in the studied population had a solitary lesion. All the multiple lesions were of pyogenic origin.

Table 14: Table showing the distribution of patients in the studied population based on the aetiology of pyogenic abscess.

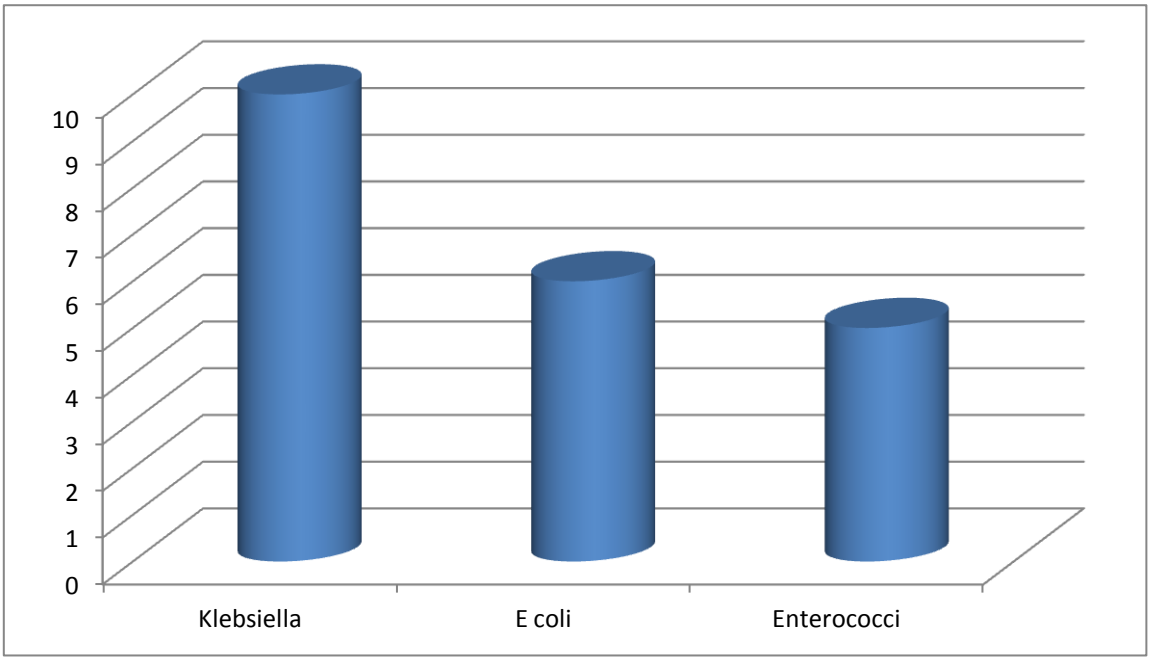
Aetiology	Number	Percentage
Biliary	9	25
Sepsis	1	2.8%
Cryptogenic	26	72.2%
Total	**36**	**100**



72.2 % of the pyogenic abscess was of cryptogenic origin. Biliary origin of abscess was present in 25 % of cases.

Table 15: Table showing the bacteria isolated from blood culture in patients with liver abscess.

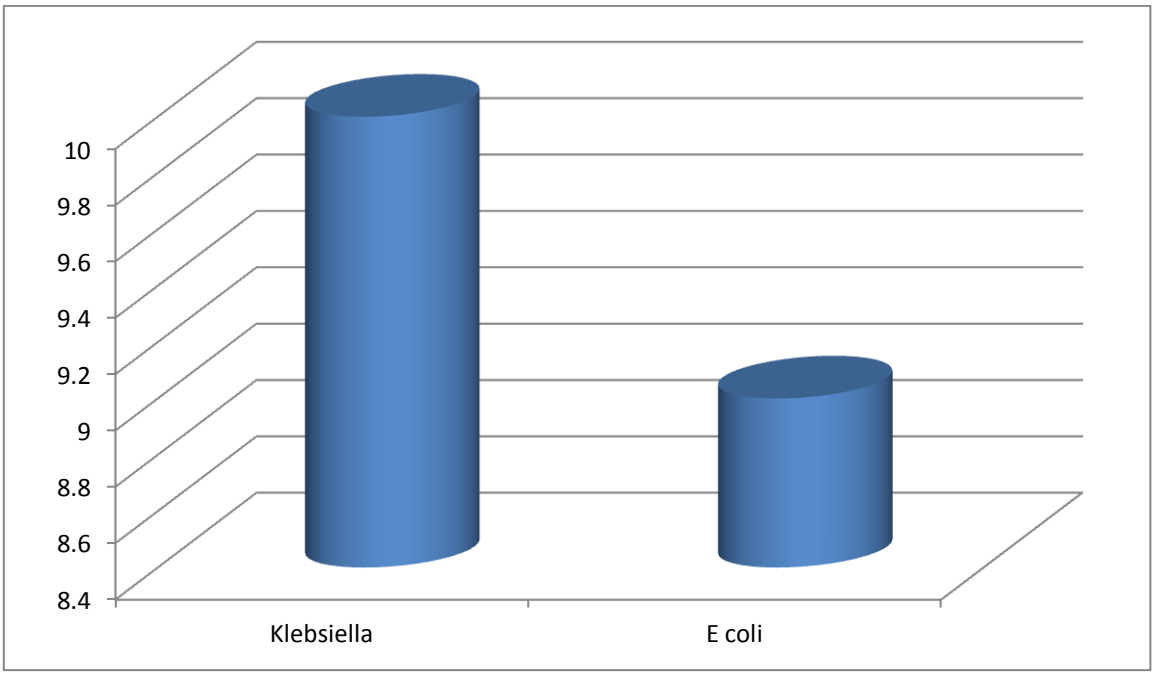
Bacteria	Number	Percentage
Klebsiella	10	47.6
E coli	6	28.6
Enterococci	5	23.8
Total	**21**	**100**



Blood culture was positive in 58.3 % of pyogenic abscess. Klebsiella was grown in 47.6 % of cases. E. coli was grown in 28.6 % of cases. Enterococci were isolated in 23.8 % of cases.

Table 16: Table showing the bacteria isolated from the culture of pus from patients with pyogenic liver abscess.

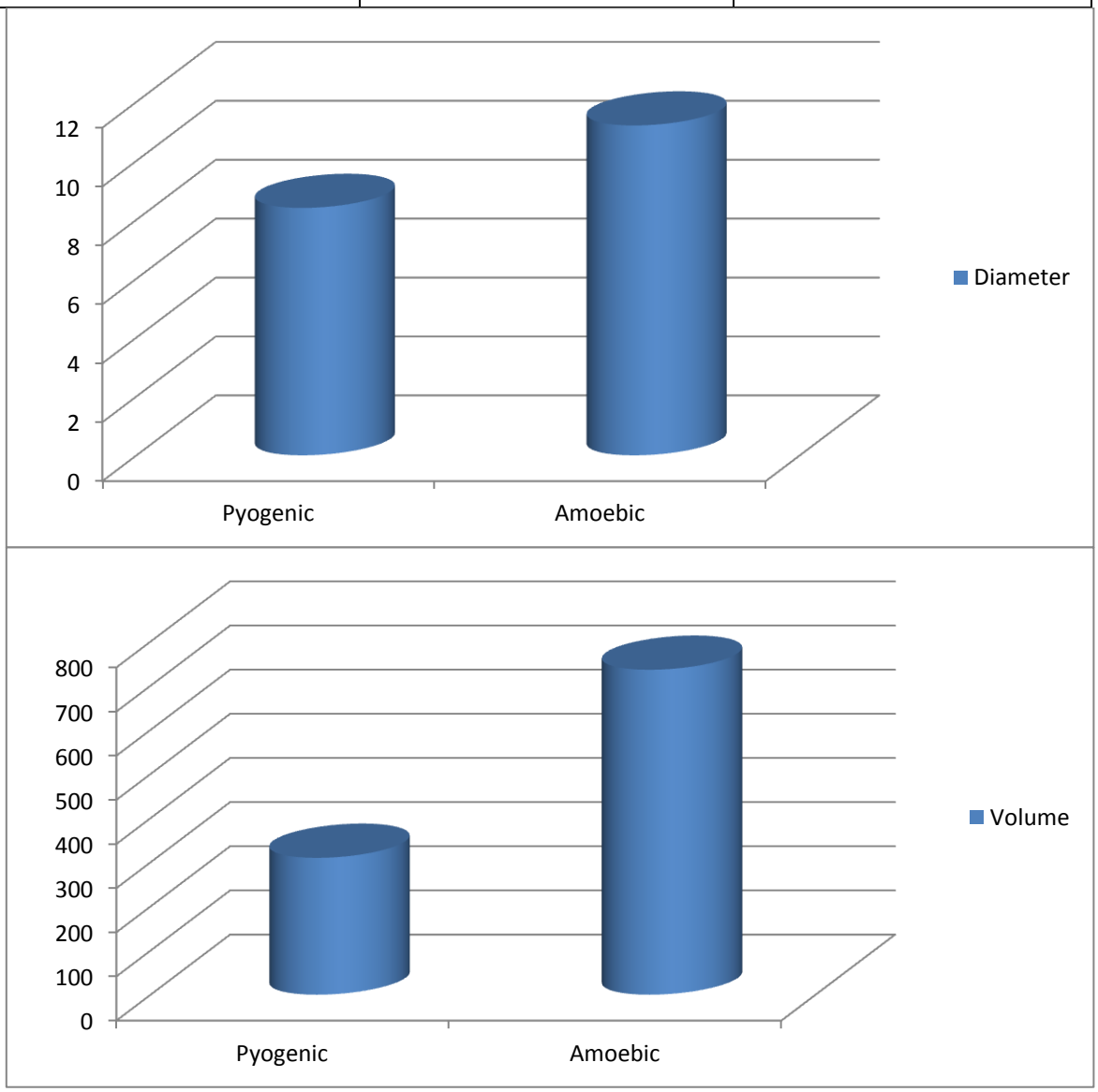
Bacteria	Number	Percentage
Klebsiella	10	52.6
E coli	9	47.4
Total	**19**	**100**



Pus culture yielded growth in 52.8 % of pyogenic abscess. Klebsiella was the predominant organism, and was present in 52.6 % of cultures. E coli constituted 47.4 % of the cultures.

Table 17: Table showing the average size and volume of the abscess cavity based on the type of liver abscess.

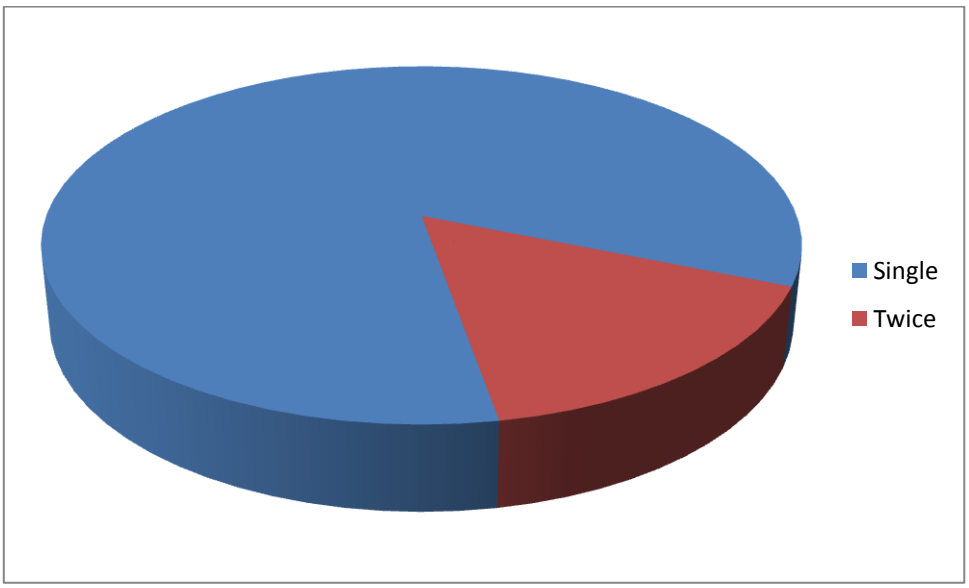
	Diameter	Volume
Pyogenic	8.4	310
Amoebic	11.2	736



Amoebic abscesses were larger with mean diameter of 11.2 cm to pyogenic abscesses with mean diameter 8.4 cm. The average volume of amoebic abscesses was 736 ml compared to 310 ml of pyogenic liver abscess.

Table 18: Table showing the number of aspirations required in successful treatment of liver abscess.

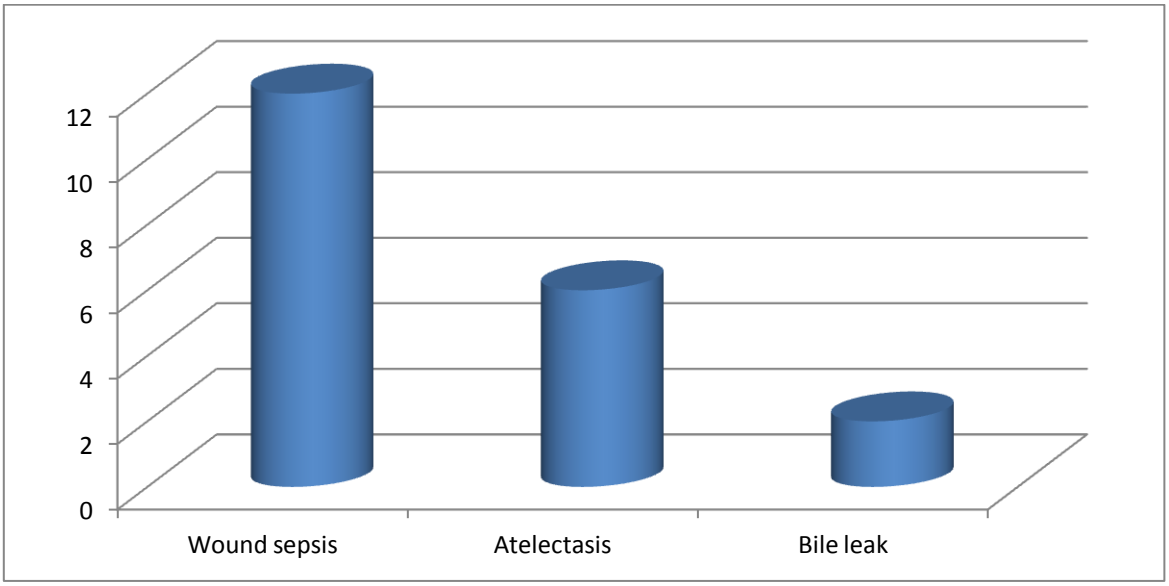
	Number	Percentage
Single	21	84
Twice	4	16



Percutaneous aspiration was successful in all the patients employed where 84 % were treated with a single aspiration and 16 % required aspiration twice.

Table 19: Table showing the common complications encountered in patients who underwent open surgical drainage of liver abscess.

Complications	Number	Percentage
Wound sepsis	12	48 %
Atelectasis	6	24 %
Bile leak	2	8 %
Total	**19**	



76 % of the patients who underwent open surgical drainage developed complications. Wound sepsis (superficial surgical site infection) was encountered in 48 % patients who underwent open surgical drainage. Right basal atelectasis was seen in 24 % of cases and 8 % cases had postoperative bile leak.

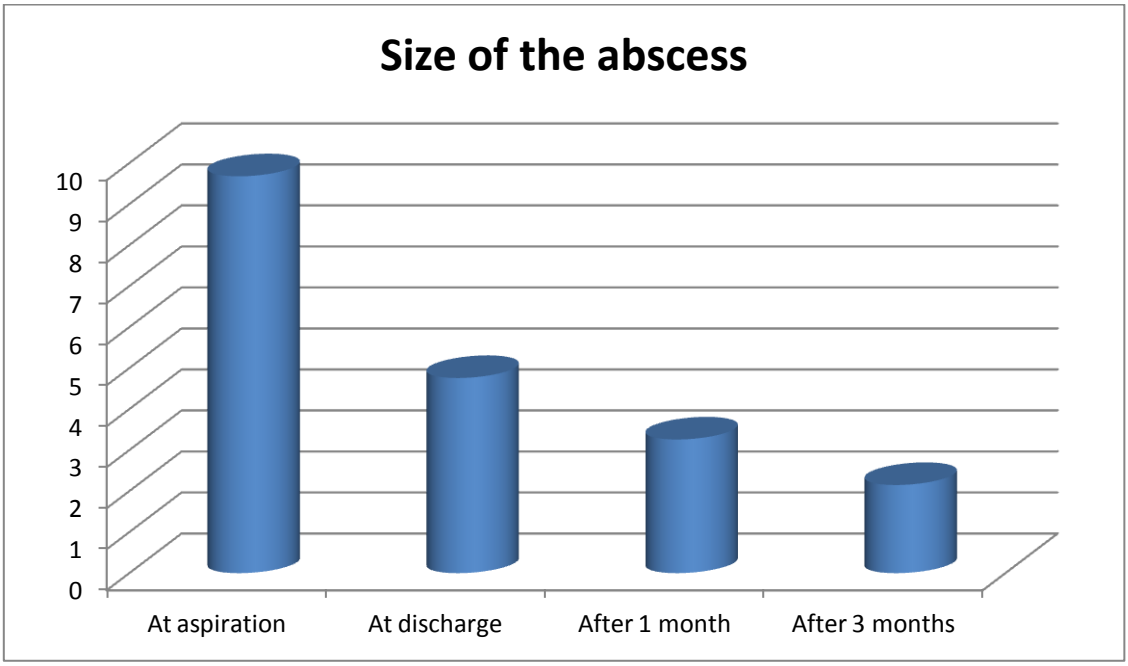
Table 20: Table showing the mean values of the investigations done in patients with pyogenic and amoebic liver abscess.

Investigations	Pyogenic liver abscess	S.D	Amoebic liver abscess	S.D
Hemoglobin	9.5	0.69	9.5	0.93
White Blood Cell count	11.7×10^3	1483	12.3×10^3	1793
Urea	38.6	8.73	37	8.7
Creatinine	0.9	0.18	0.9	0.2
Total Bilirubin	2	0.8	2.23	0.68
SAP	103.4	26.58	119.1	34.6
ALT	49.8	12.72	89.1	23.72
AST	58.2	13.20	89.7	15.94
Albumin	3.3	0.35	3.1	0.34
Prothrombin time	15.4	2.73	15.8	2.42

Patients with amoebic liver abscess had a marginal increase in mean values of WBC count compared to pyogenic liver abscess. The mean values of serum bilirubin, serum alkaline phosphatase, AST and ALT were higher in patients with amoebic liver abscess compared to pyogenic liver abscess.

Table 21: Table showing the average size of liver abscess following aspiration and at follow up.

	Size of Abscess	Percentage
At Aspiration	9.68 cm	**100**
At Discharge	4.76 cm	**49.2**
At 1 month of follow up	3.26 cm	**33.7**
At 3 months of follow up	2.15 cm	**22.2**

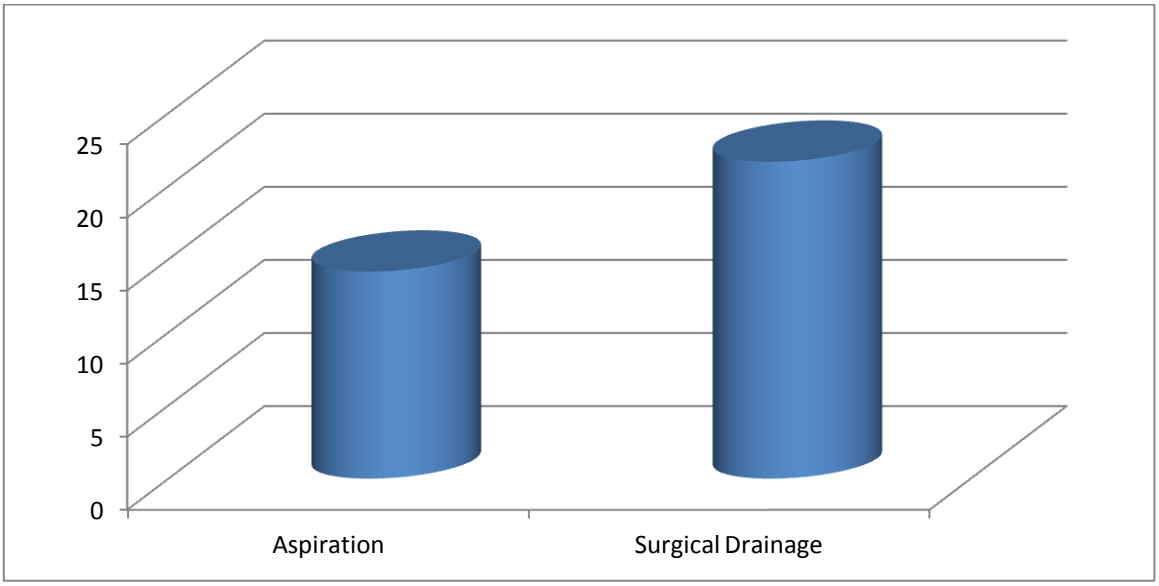


There was a 50 % reduction in the size of abscess following needle aspiration at discharge. Most of the patients had a residual abscess cavity at 3 months of follow up.

Table 22: Table showing the average duration of stay in patients who underwent percutaneous aspiration and open surgical drainage.

	Aspiration		Open Surgical Drainage	
Variable	Mean	Std. Dev.	Mean	Std. Dev.
Duration of stay	14.16	3.21	21.68	2.36

P = 0.03



The average duration of stay was longer in patients who underwent open surgical drainage compared to percutaneous aspiration. The p value was < 0.05 and hence the result was statistically significant.

Discussion

Analysis of age and sex distribution.

Majority of the patients were between the ages of 26 to 65 years. The mean age of the studied population was 44.8 years. Males constituted 92 % of the studied population. These results are in concordance with previous studies.

Studies	Mean age
Giorgio et al	45.3 years (n = 115)
Torres OJM et al[44]	45.2 years (n = 21)
Rajak et al	35.5 years (n = 50)
Present study	44.8 years (n = 50)

Studies	Male	Female
Giorgio et al	51.3 %	48.7 %
Torres OJM et al	61.9 %	38.1 %
Rajak et al	76 %	24 %
Shyam mathur et al	96 %	4 %
Present study	92 %	8 %

Present study shows a high prevalence of liver abscess in males. This is also appreciated in other studies like Rajak et al and Shyam mathur et al. The prevalence of liver abscess seems to be equal among the sexes in studies outside India.

Analysis of symptoms and signs.

Pyogenic liver abscess

Symptoms and signs	Malik et al[45]	Yoo HM et al[46]	Present study
Abdominal Pain	62%	84%	97.2 %
Fever	91%	75%	77.8 %
Diarrhoea	10%	13%	11.1 %
Jaundice	34%	9%	8.3 %
Hepatomegaly	65%	42%	41.7 %
Abdominal tenderness	70%	88%	58.3 %

Amoebic liver abscess

Symptoms and signs	Yoo HM et al	Hayat et al[47]	Present study
Abdominal Pain	90%	94%	78.6 %
Fever	63%	78%	78.6 %
Diarrhoea	15%	18%	28.6 %
Jaundice	2%	10%	12 %
Hepatomegaly	40%	48%	57.1 %
Abdominal tenderness	88%	94%	64.3 %

Right upper quadrant pain, fever and abdominal tenderness (right lower intercostal tenderness or epigastric tenderness) were the three most consistent findings in pyogenic liver abscess. Diarrhoea and jaundice were found in about 10% of the patients. In amoebic liver abscess, abdominal pain and fever were the most consistent findings. Hepatomegaly was more common in amoebic liver abscess probably due to the presence of larger abscesses compared to pyogenic group.

Alcohol consumption was seen in 70% of the patients. Diabetes mellitus was prevalent in 10 % of the patients.

Interpretation of Investigations.

Pyogenic liver abscess

Investigations	Alexopoulou et al[48]	Yoo HM et al	Present study
Hemoglobin < 9g/dl	-	25 %	33.3 %
Leukocytosis	81.8 %	81 %	77.8 %
T Bilirubin >2 mg/dl	24.2 %	25 %	30. 5 %
AST >60	39.4 %	25 %	38.9 %
ALT >60	-	25 %	25 %
SAP >115	45.4 %	86 %	30.6
ALB <3	45.4 %	49 %	27.8

Leukocytosis was seen in 77.8 % of patients with pyogenic liver abscess. None of the other laboratory parameters were consistent with all patients. Amoebic liver abscesses also had associated leukocytosis. Elevation of AST and ALT was seen in more than 80 % patients of amoebic liver abscess, probably due to prolonged duration of the disease.

Amoebic liver abscess

Investigations	Yoo HM et al	Present study
Hemoglobin < 9g/dl	17 %	28.6 %
Leukocytosis	88 %	71.4 %
T Bilirubin >2	10 %	21.4 %
AST >60	11 %	92.8 %
ALT >60	10 %	85.7 %
SAP >115	76 %	42.8 %
ALB <3	71 %	28.6 %

Analysis of type of abscess.

	Rajak et al	Torres OJM et al	Present study
Pyogenic	22 %	57.1 %	72
Amoebic	40 %	42.9 %	28
Indeterminate	38 %	-	-

Pyogenic abscess was more common than amoebic abscess in the studied population. Amoebic abscesses were common in the studies like Torres et al and Rajak et al which were conducted in the tropical countries. But the overall higher prevalence of pyogenic liver abscesses compared to the amoebic liver abscess in our study concur with other studies mentioned.

Analysis of the lobe of involvement of abscess.

	Rajak et al	J. Rahimian[49]	A. Alexopoulou et al	Present study
Right	68 %	70.5 %	69.7 %	**86 %**
Left	14 %	14.1 %	12.1 %	**8 %**
Bilobar	18 %	5.1 %	18.2 %	**6 %**

Liver abscess was common on the right lobe of liver. Left lobe abscess and bilobar involvement was seen in less than 10 % of cases.

Analysis of the number of abscesses.

	Rajak et al	Torres OJM et al	A. Alexopoulou et al	Present study
Solitary	76 %	47.6 %	75.7 %	90 %
Multiple	24 %	52.4 %	24.3 %	10 %

Liver abscess tend to be solitary in our studied population as the majority of the liver abscess were cryptogenic. Multiple abscesses are more a feature of liver abscess which arise from a biliary aetiology. These findings are in concordance with studies of Rajak et al and A. Alexopoulou et al.

Analysis of aetiology of pyogenic liver abscess.

	J. Rahimian et al	Yoo HM et al	Present study
Biliary	43 %	51 %	25
Sepsis	5 %	3 %	2.8%
Cryptogenic	48.1 %	38 %	72.2%

Biliary sepsis like cholangitis was the predominant aetiological factor for pyogenic liver abscess in most of the studies. In our study, the predominant cause of pyogenic liver abscess remained cryptogenic. Cryptogenic abscesses occur in 10 – 45 % of the patients, depending on the extent of investigations used to identify the cause. [14] The patients in our study were not keen on pursuing further investigations as they became symptomatically better; the number of patients in the cryptogenic group remained high.

Analysis of blood culture results.

	S.C. Chen et al[50]	Khee-Siang Chan[51]	Present study
Klebsiella sp	79.3 %	93.4 %	47.6
E coli	12.3 %	6.6 %	28.6
Enterococci	2 %	-	23.8

Blood culture from patients with pyogenic liver abscess yield a positive growth in about 50 – 60 % of cases. In our studied population 62 % of the patients with pyogenic abscess had a positive growth on culture. The most common organism isolated was klebsiella sp. The results concur with other studies like S. C. Chen et al and Khee-Siang Chan et al. Anaerobic cultures were not done in the study.

Analysis of pus culture results.

	Yoo HM et al	K.L. Goh et al[52]	Present Study
Klebsiella	24 %	25 %	52.6
E coli	48.6 %	33.3 %	47.4
Enterococci	10. 8 %	12.5 %	-

In contrast to blood cultures, pus from the abscess cavity yield growth in about 80 – 97 % of patients. [14] But in our study pus from the abscess

cavity yielded growth in 60 % of the patients. Klebsiella was the most common organism cultured. In a few patients there was a difference in organism cultured from the abscess cavity and the blood of the same individual. This might be due to polymicrobial nature of liver abscess or due to administration of antibiotics prior to abscess drainage which might alter the pus culture results. Anaerobic cultures were not done in the study.

Analysis of treatment.

Percutaneous aspiration of the abscess was successful in all the patients. 16 % of the patients required a second aspiration as the clinical improvement was marginal and significant amount of the residual abscess was present. Rajak et al had only 60 % success in their percutaneous aspiration group, while other studies had reported almost 100 % cure with minimal or no complications.

	Rajak et al	Giorgio et al	Present study
Single	44 %	92.3 %	84
Twice	16 %	7.7 %	16

Open surgical drainage was also successful in all the patients. But complications were encountered in 76 % of the patients ranging from local wound sepsis and self limiting bile leak.

The duration of hospital stay was longer in patients who underwent open surgical drainage, partly due to the post operative complications that ensued.

The patients were on follow up for a period of 3 months. None of the patients had relapses or recurrences. 8 patients of the aspiration group did not come for follow up at the end of 3 months. Of the remaining 17 patients, 4 patients had no demonstrable abscess cavity, while the other patients had a 78 % reduction in the size of the abscess cavity. As clinical improvement was considered as the end point of therapy, no further procedures were undertaken to these patients. Patients in the open surgical drainage group had no demonstrable abscess cavity at discharge.

Conclusion

Liver abscess occurred most commonly in age group of 26 to 65 years. Liver abscess was more common in males. Abdominal pain and fever were the most common presenting symptoms. Tachycardia and right intercostal tenderness were the common signs present in liver abscess. Leukocytosis was most common laboratory finding. Elevation of AST and ALT were the most common abnormality seen on liver function tests.

Pyogenic liver abscess was the common type of liver abscess. Most of the abscesses were solitary and involved the right lobe of liver.

Klebsiella species were the most common organisms isolated from blood culture and the pus culture. The cause of liver abscess remained obscure in most of the patients.

Percutaneous needle aspiration is a safe interventional modality and was successful in all the patients. Open surgical drainage was also effective in all the patients employed. Surgical drainage was associated with post operative complications in 76 % of patients.

Percutaneous needle aspiration is a better first line management in the treatment of liver abscess. Surgical drainage can be reserved for patients in whom percutaneous needle aspiration is unsuccessful.

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A COMPARATIVE STUDY OF OPEN SURGICAL DRAINAGE AND EARLY PERCUTANEOUS NEEDLE ASPIRATION OF LIVER ABSCESS.

PROFORMA

Patient's Name : Age : Sex :
Occupation : Socio economic status :
Address : I.P No:

D.O.A. :
D.O.D. : Duration of stay in Hospital :

CHIEF COMPLAINTS :

HISTORY OF PRESENTING ILLNESS :

Symptoms
Abdominal pain : Yes / No
- Duration :
- Site :
- Character :
- Radiation :

Fever : Yes / No
- Duration :
- Associated with chills and rigors :

Diarrhoea / Dysentery :
- Duration :
- Mucous diarrhoea :
- Blood in stools :

Vomiting :
- Duration / Frequency :
- Bilious :

Jaundice : Yes / No
Cough : Yes / No
Distension of Abdomen : Yes / No
Altered Sensorium : Yes / No
Any other :

PAST HISTORY :
Liver Abscess: Yes / No
Gallstone disease: Yes / No
Diarrhea : Yes / No
Jaundice : Yes / No
Tuberculosis: Yes / No
Surgery : Yes / No Comment:
Trauma : Yes / No Comment:

FAMILY HISTORY :

PERSONAL HISTORY :
Diabetes Mellitus: Yes / No Duration:
Hypertension: Yes / No Duration:

ALCOHOL CONSUMPTION : Yes / No
Duration :
Type : Local Arrack: Yes / No
Amount/Frequency:

SMOKING : Yes / No

Appetite :
Sleep :
Bowel and Bladder :

TREATMENT HISTORY :

GENERAL PHYSICAL EXAMINATION :

Build :
Nourishment :

Pallor : Jaundice : Cyanosis :
Clubbing : Lymphadenopathy : Edema:
Pulse : /min
B.P. mm of Hg
Respiration : per minute
Temperature :

SYSTEMIC EXAMINATION :
ABDOMEN :

Shape :
Umbilicus :
Movements :
Tenderness : Present / Absent
Rigidity : Present / Absent
Liver : Palpable / non palpable Liver span : cm
Surface :
Right intercostal tenderness: Present / Absent
Spleen : Palpable / non palpable
Scrotum / Testes :
External Hernial Orifices :
Free fluid:
Bowel sounds : Present / Absent

Per Rectal Examination :

RESPIRATORY SYSTEM :

CARDIOVASCULAR SYSTEM :

CENTRAL NERVOUS SYSTEM :

INVESTIGATIONS :

Blood :
Group and Type:
Hb : gm%
T.C. : c/cmm
D.C.: N %, L %, E%, M%, B%
ESR : mm/hr
BT :
CT :
Prothrombin time : sec INR:

RBS : mg/dl
Blood urea : mg/dl
S creatinine : mg/dl

Urine :
Albumin : Yes / No
Sugar : Yes / No
Microscopy :

Stool :

Ova / cyst :
Occult blood : Yes / No

SPECIAL INVESTIGATIONS :

Liver Function Tests :

Bilirubin :	ALP:	Total proteins :
Direct:	ALT:	Albumin:
Indirect:	SAP:	Globulin:
PT :		A:G ratio :

Blood Culture:

HIV I AND II (ELISA) :

HBsAg:

RADIOLOGICAL INVESTIGATIONS :

Plain X-ray Chest PA View :

Plain X-ray abdomen erect ;

ULTRASONOGRAPHY ABDOMEN :

Lobe of Involvement:
Size:
Ascites: Yes / No
Other Pathology:

CT ABDOMEN:

Lobe of Involvement:
Size:
Ascites: Yes / No
Other Pathology:

OTHER INVESTIGATIONS :

DIAGNOSIS :

TREATMENT :

Percutaneous aspiration / Surgery.

COMMENTS :

A COMPARATIVE STUDY OF OPEN SURGICAL DRAINAGE AND EARLY PERCUTANEOUS NEEDLE ASPIRATION OF LIVER ABSCESS.

Proforma for percutaneous Aspiration.

Patient Name: Age: Sex:
I. P. No:

DOA: DOAspiration: DOD:

Image Guidance: USG / CT

Amount Aspirated:
Type: Amoebic / Pyogenic

Pus Culture:
Microscopy:

Complications:

USG 1 week After Aspiration:
Size:

Re-aspiration if done: Yes / No
Comment:

Followup USG:

Complications:

Follow up Investigations:

LFT:
RFT:

A COMPARATIVE STUDY OF OPEN SURGICAL DRAINAGE AND EARLY PERCUTANEOUS NEEDLE ASPIRATION OF LIVER ABSCESS.

Proforma for surgical drainage:

Patient Name: Age: Sex:
I. P. No:

D O A: D O Surgery: D O D:

Incision: Anaesthesia:

Drainage amount:
Pus Type: Amoebic / pyogenic
Drain used:

Post operative period:

Pus Culture:

Pus Microscopy:

Complications:

Followup USG:

Followup Investigations:

LFT:
RFT:

Comment:

MASTER CHART

Si no	NAME	IP NO	AGE	SEX	DURATION OF STAY	ABDOMINAL PAIN	FEVER	DIARRHOEA	JAUNDICE	ALCOHOL	DM	HEPATOMEGALY	IC tenderness	PULSE	Hb	TC x 1000	Urea	Creat	TB	DB	IB	SAP	ALT	AST	ALB	PT	BLOOD CULTURE
1	ELUMALAI	90273	65	m	12	y				y		y	y	94	9.8	10.8	34	1	2	0.6	1.4	76	34	45	3.6	13	
2	DAMODHARAN	374	42	m	14	y	y			y	y		y	98	9	12	40	1	2.2	0.8	1.4	120	40	48	3	12	
3	KUPPAN	932	48	m	11	y	y					y	y	92	9.2	13	36	0.8	1.6	0.6	1	96	45	54	3.4	14	kleb
4	ARAIMMAL	4859	65	f	25	y	y						y	104	10	11.4	45	0.9	2.4	1	1.4	136	46	58	4	12	kleb
5	PARAMASIVAN	7302	60	m	22	y				y			y	98	8.8	9.7	34	0.7	1.8	0.4	1.4	84	53	60	2.8	13	e coli
6	BOOPALAN	7684	42	m	20	y				y				112	8.6	8.4	36	0.8	1.8	0.6	1.2	92	67	58	3	12	
7	PERIASWAMY	8575	56	m	16	y	y			y		y	y	104	10.2	12.6	38	1	2	0.8	1.2	104	34	42	3.5	14	ent
8	CHELLAN	9344	60	m	18	y	y			y			y	99	9	11.4	45	1.1	2.4	0.9	1.5	124	42	48	3.4	12	kleb
9	MUTHU	9539	55	m	22	y						y		106	9.6	14	42	1	2.8	1.2	1.6	146	87	92	3	13	
10	PONNAMMAL	14961	46	f	23	y							y	98	8.8	9.8	42	0.9	1.3	0.4	0.9	96	35	54	2.8	12	ent
11	ELANGOVAN	34578	50	m	13	y	y	y		y	y	y		108	9	13.8	41	0.9	2.8	1.8	1	154	125	99	2.8	14	
12	SIVAKUMAR	45162	32	m	9	y	y		y	y		y		114	8.6	12.8	54	1.4	3.8	1.6	2.2	168	112	104	2.8	14	
13	MAHESH	45426	26	m	19	y	y			y			y	110	9.2	8.4	49	1.3	2.1	0.9	1.2	122	64	68	3	15	
14	ETTIAPPAN	49384	34	m	15	y	y			y				96	9.8	11.8	32	0.7	1.8	0.6	1.2	98	47	54	4	21	kleb
15	SUBRAMANI	74817	45	m	21	y								98	9.6	9.9	28	0.7	2	0.6	1.4	94	88	94	3.2	18	
16	ARJUNAN	67	28	m	17		y			y		y	y	96	9.8	10.9	25	0.6	2.4	0.5	1.9	112	94	92	3.5	16	
17	KUPPUSWAMY	149	22	m	24	y	y			y				102	10	12.5	42	1	1.4	0.4	1	84	46	53	3.6	19	ent
18	VENUGOPAL	675	62	m	23	y				y		y	y	104	8.8	13	44	1.1	2.6	0.6	2	108	78	85	3	17	
19	GOPAL	830	58	m	16	y	y			y			y	93	9	13.4	22	0.7	1.2	0.5	0.7	104	46	52	3	16	
20	VEERASWAMY	1845	49	m	20	y	y	y		y	y	y		96	9.2	12.6	43	1	2.1	0.7	1.4	98	31	46	3.1	19	e coli
21	CHANDRAMMAL	2350	36	m	28	y	y		y	y		y	y	102	9.2	10.8	56	1.4	3.6	1.6	2	162	28	38	3.2	15	kleb
22	CHOKKAMMAL	2673	57	f	24		y						y	92	8.8	9.4	48	1	2.2	0.7	1.5	88	73	84	3	14	kleb
23	PERUMAL	4360	61	m	22	y	y	y		y				100	8	8.3	38	1	2	0.6	1.4	84	87	92	2.6	12	
24	VENKATESAN	4629	34	m	21	y	y	y	y				y	104	9	11.8	54	1.1	4	1.6	2.4	188	54	66	2.8	13	e coli
25	MURUGAN	5931	56	m	21	y				y		y		90	9.4	13.4	38	0.9	1.8	0.8	1	82	38	54	3	16	
26	RAJESH	6028	29	m	13	y	y			y			y	84	9.6	14.2	38	0.8	1.6	0.7	0.9	65	46	58	3.2	18	
27	KARUNAKARAN	6437	34	m	13	y	y	y		y		y	y	98	9.6	11.4	24	0.7	2.4	0.9	1.5	122	98	100	3.2	16	
28	BASKAR	7394	38	m	20	y				y		y	y	92	9.8	11.2	36	0.9	1.4	0.6	0.8	72	46	49	3.5	15	
29	BALAN	8142	45	m	21	y	y							103	10.2	9.8	24	0.7	1.8	0.5	1.3	78	58	62	3.8	18	kleb
30	MUNISWAMY	8353	57	m	25	y	y			y		y		116	10	12.2	38	1	1	0.2	0.8	80	56	63	4	22	
31	LOGANATHAN	8503	63	m	22	y	y			y			y	102	8	12.8	40	1	2.6	0.8	1.8	86	102	98	2.8	14	
32	MUNIAMMAL	9148	31	f	19	y	y					y		98	9.8	11.6	34	0.9	1.4	0.4	1	88	65	72	3.1	15	e coli
33	MUTHUKUMAR	9573	44	m	12	y	y						y	108	8.2	11.4	28	0.8	1.6	0.5	1.1	94	57	62	2.8	14	
34	KUMAR	11672	64	m	15	y	y			y				96	9	13.4	30	1	2.8	1	1.8	132	44	54	3.4	14	ent

35	APPADORAI	13591	59	m	14	y		y		y	y	y	y	108	9.2	12.8	48	1.2	1	0.3	0.7	64	72	84	3	13	
36	NATARAJ	26271	29	m	12	y	y						y	112	10	13.2	35	0.8	1.6	0.3	1.3	78	92	88	3.5	18	
37	KRISHNAN	28556	42	m	16	y	y		y	y		y		96	9.6	11.8	56	1	3.6	1.4	2.2	144	58	53	3.2	17	kleb
38	SHEKAR	33524	37	m	14		y			y			y	92	10	14.2	47	0.8	2.1	0.9	1.2	184	120	114	3.8	15	
39	CHINNAPAIYAN	35292	46	m	19	y	y		y	y		y		90	10.2	14	25	0.7	4.2	1	3.2	92	64	78	3.4	14	ent
40	ELUMALAI	36993	45	m	21	y	y						y	110	10	14	31	0.7	1.6	0.7	0.9	102	59	65	3.5	17	e coli
41	BABU	37230	36	m	11	y	y			y		y		98	10.2	11.2	42	1	2.1	0.8	1.3	118	64	84	3.6	19	
42	KASIYAMMAL	43489	54	f	22	y	y	y					y	104	9	10.4	29	0.9	1.4	0.6	0.8	96	42	36	3	18	
43	PONNUSWAMY	46425	52	m	20	y	y				y	y	y	96	9.8	12.6	36	1	1.2	0.5	0.7	128	62	56	3.2	14	kleb
44	PARTHIBAN	60273	28	m	9	y				y				112	12	11	52	1.2	3	1.4	1.6	110	36	64	3.1	18	
45	ALAGESAN	65833	34	m	15		y		y	y		y	y	88	11	10.4	40	0.9	1.4	0.8	0.6	145	72	78	3	21	
46	BOOPATHI	68564	38	m	23	y	y			y				94	10.2	13	38	0.8	1.5	0.7	0.8	98	42	54	3.4	15	kleb
47	KESAVAN	73466	29	m	12	y	y	y				y	y	104	9.8	12.8	28	0.7	1.2	0.6	0.6	84	38	48	3.2	18	
48	KADIRESAN	79230	31	m	19	y	y			y			y	96	11	14	34	1	1.6	0.6	1	102	54	72	3.5	15	
49	RANGANATHAN	83482	52	m	20	y	y			y		y	y	84	9	12	41	1.2	2	0.8	1.2	118	69	84	3.8	19	e coli
50	PRABHU	98263	34	m	13	y	y			y				98	10	11	30	1	1.4	0.8	0.6	88	31	34	3.2	13	

Continued on next page...

MASTER CHART (continued)

Si No	Name	IP No	LOBE	SIZE	SOL/MUL	PYO/AMOE	Treatment	No of Asp	cause	pus c/s	Complications	At discharge	At 1 month	At 3 months
1	ELUMALAI	90273	R	8	S	P	Asp	1	crypt		Nil	6	4	2
2	DAMODHARAN	374	R	10	S	P	Asp	1	crypt	e coli	Nil	4	dnf	dnf
3	KUPPAN	932	R	9	S	P	Asp	1	crypt	kleb	Nil	5	dnf	dnf
4	ARAIMMAL	4859	R	8	S	P	Sur		biliary	kleb	A			
5	PARAMASIVAN	7302	R	8	M	P	Sur		crypt	e coli	W + A			
6	BOOPALAN	7684	L	6	S	P	Sur		crypt	e coli	Nil			
7	PERIASWAMY	8575	R	8	S	P	Asp	1	crypt		Nil	6	4	3
8	CHELLAN	9344	R	8	S	P	Sur		biliary	e coli	W			
9	MUTHU	9539	R	12	S	A	Sur		amoebic		W + A			
10	PONNAMMAL	14961	R	9	S	P	Sur		crypt		NIL			
11	ELANGOVAN	34578	R	11	S	A	Asp	1	amoebic		Nil	5	3	2
12	SIVAKUMAR	45162	R	12	S	A	Asp	1	amoebic		Nil	4	3	no cavity
13	MAHESH	45426	R	9	S	P	Sur		biliary	kleb	W			
14	ETTIAPPAN	49384	R	8	S	P	Asp	1	crypt	kleb	Nil	6	5	3
15	SUBRAMANI	74817	R	10	S	A	Sur		amoebic		NIL			
16	ARJUNAN	67	R	13	S	A	Asp	1	amoebic		Nil	4	2	2
17	KUPPUSWAMY	149	L	8	S	P	Sur		crypt		W			
18	VENUGOPAL	675	R	10	S	A	Sur		amoebic		NIL			
19	GOPAL	830	R	10	S	P	Asp	1	crypt	e coli	Nil	6	4	dnf
20	VEERASWAMY	1845	R	9	S	P	Asp	1	biliary	e coli	Nil	6	5	dnf
21	CHANDRAMMAL	2350	R	8	S	P	Sur		biliary		A + B			
22	CHOKKAMMAL	2673	B	8	M	P	Sur		sepsis	kleb	W			
23	PERUMAL	4360	L	8	S	A	Sur		amoebic		NIL			
24	VENKATESAN	4629	R	9	S	P	Asp	2	biliary	e coli	Nil	4	dnf	dnf
25	MURUGAN	5931	R	8	S	P	Sur		crypt		W + A			
26	RAJESH	6028	R	10	S	P	Asp	1	crypt	kleb	Nil	5	3	2
27	KARUNAKARAN	6437	R	12	S	A	Asp	1	amoebic		Nil	6	4	2
28	BASKAR	7394	R	9	M	P	Sur		biliary		B			
29	BALAN	8142	R	8	S	P	Sur		crypt		W			
30	MUNISWAMY	8353	R	8	S	P	Sur		crypt	kleb	A			
31	LOGANATHAN	8503	R	11	S	A	Sur		amoebic		W			
32	MUNIAMMAL	9148	R	7	S	P	Sur		crypt		NIL			
33	MUTHUKUMAR	9573	R	8	S	P	Asp	1	crypt	kleb	Nil	3	3	no cavity
34	KUMAR	11672	R	10	S	P	Asp	1	biliary	e coli	Nil	4	3	2
35	APPADORAI	13591	B	8	M	P	Asp	2	crypt		Nil	5	dnf	dnf
36	NATARAJ	26271	R	13	S	A	Asp	1	amoebic		Nil	6	4	2
37	KRISHNAN	28556	R	7	S	P	Asp	1	crypt	kleb	Nil	4	2	no cavity
38	SHEKAR	33524	R	12	S	A	Asp	1	amoebic		Nil	3	dnf	dnf
39	CHINNAPAIYAN	35292	R	9	S	P	Sur		crypt		W			

40	ELUMALAI	36993	R	8	S	P	Asp	2	crypt		Nil	5	3	2
41	BABU	37230	R	8		P	Asp		crypt	-	Nil	4	3	2
42	KASIYAMMAL	43489	R	9		P	Sur		crypt	-	NIL			
43	PONNUSWAMY	46425	R	8		P	Sur		crypt	-	NIL			
44	PARTHIBAN	60273	L	10		P	Asp		crypt	-	Nil	5	3	2
45	ALAGESAN	65833	R	10		A	Asp		amoebic	-	Nil	5	dnf	dnf
46	BOOPATHI	68564	R	9		P	Sur		crypt	kleb	W			
47	KESAVAN	73466	R	11		A	Asp		amoebic	-	Nil	4	2	2
48	KADIRESAN	79230	R	12		A	Sur		amoebic	-	NIL			
49	RANGANATHAN	83482	B	8		P	Sur		biliary	e coli	W			
50	PRABHU	98263	R	8		P	Asp		crypt	-	Nil	4	2	no cavity